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MEDICAL AND SURGICAL ASPECTS OF GOITER.*

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Baumann in 1895 discovered that large amounts of iodin were normally present in the thyroid gland and that there was only a small amount of iodin in goitrous glands. This was a suggestion at least that treatment with iodin might be beneficial to patients suffering from goiter. Chatin's observation that goiters were more prevalent in regions where the iodin content was low was also at least presumptive evidence that this substance might in some way be used in the treatment of enlargement of the thyroid gland. Kendall showed that the maximal amount of iodin which could be stored in the normal thyroid gland was about 6 mg. for each grain of dry gland; that the normal was about 2 mg. for each grain of dry gland, and that in goitrous glands it was so low as to be undetectable. In 1914, he isolated from the thyroid gland the specific iodin containing substance, thyroxin, which contains 65 per cent of iodin. Experiments showed that 1/66 of a grain of thyroxin would cause about a 3 per cent increase in the metabolic rate and that a single dose kept the rate elevated for a period of about six weeks in a person who had little or no thyroid tissue. If thyroxin or thyroid extract is administered to a person with a normal thyroid gland, the symptoms which characterize exophthalmic goiter will not be produced.

The part which the thymus plays in hyperthyroidism is not known. The study of this gland in connection with goiters is, however, extremely interesting. Boothby and Giordano found, in reviewing reports of a large number of necropsies of patients dying from exophthalmic goiter, that thymic hyperplasia existed in 66 per cent of the cases, whereas among patients with adenomatous goiters with hyperthyroidism, thymic hyperplasia was found in 50 per cent of cases.

According to Plummer, goiter may be classified as colloid or endemic, adenomatous without hyperthyroidism, adenomatous with hyperthyroidism and exophthalmic.

Diffuse colloid or endemic goiter is most frequently observed at puberty or shortly afterward. Kimball and Marine in 1917 found that of 2305 girls of the city schools of Akron, Ohio, 445 had colloid goiter. McCarrison believed that the cause of this type of goiter is a toxic substance created by the presence of an intestinal micro-organism. Plummer has suggested in addition to this that possibly the intestinal flora may appropriate part of the small amount of iodin in food, thus causing iodin deficiency.

The consensus of opinion of most observers is that this type of goiter is due to iodin insufficiency and that the colloid

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material is stored in the thyroid gland as more or less of a by-product in the gland's attempt to maintain as nearly as possible the normal thyroxin content of the body.

This type of goiter in itself does not produce hyperthyroidism. Occasionally persons who are aware of its presence become nervous and a subsequent diagnosis of exophthalmic goiter may be made. With a normal metabolic rate, exophthalmic goiter can practically be ruled out.

The administration of desiccated thyroid usually causes a marked decrease in the size of this type of goiter. Basal metabolic estimation should occasionally be made and symptoms of hyperthyroidism should be watched for during the treatment.

In Kimball and Marine's series, 2190 girls without goiter were given 30 grains of sodium iodin over a period of two weeks twice yearly and goiter developed in only five.

Occasionally colloid goiters reach a tremendous size after puberty. If obstructive respiratory symptoms develop, operation should be resorted to because not infrequently adenomas may be present and may become hyper-active following the administration of thyroxin or thyroid. Exophthalmic goiter as a condition superimposed on a colloid goiter has been seen in a few instances but is not common. The microscopic picture characterizing this type of goiter is an excess of colloid distending the acini and usually causing flattening of the parenchymal cells.

Adenomatous goiter with hyperthyroidism is rare before the twentieth year, and comparatively so until after the thirtieth year of life. It can be classified along with colloid or endemic goiter and often is referred to as non-toxic goiter because symptoms of hyperthyroidism are absent. In this type of goiter there is apparently no alteration in the thyroxin content of the body. The geographic distribution is about the same as that of colloid goiter; usually it is first noticed during the adolescent period, and consequently adenomatous goiter without hyperthyroidism, and colloid goiter are thought to have the same etiology. The formation of adenomas is thought to be a compensatory development of the thyroid gland, to supply the demands of the body for thyroxin. The prevention of adenomatous goiter, as of colloid goiter, is thought to be possible in most instances.

The adenomas may show cystic, calcareous, or hemorrhagic degeneration, may vary in size, and may be single or multiple. Microscopic examination in this type of goiter may disclose evidence of intra-adenomatous or of extra-adenomatous hypertrophy, like that found in exopthalmic goiter, but this does not necessarily mean that symptoms of hyperthyroidism are present.

It has been observed, however, that after the age of forty there is a tendency for adenomatous goiter to become hyperactive. The administration of compounds of iodin to persons more than thirty years of age with adenomatous goiter may cause hyperthyroidism. Adenomatous goiters may reach an enormous size, causing marked respiratory embarrassment and occasionally partial unilateral or bilateral paralysis of the vocal cords. The treatment is surgical. The possibility of malignancy developing in this type of goiter is an added indication for surgical intervention.

A careful survey of cases of adenomatous goiter with hyperthyroidism shows that a large percentage occurs after forty years of age. It is comparatively rare before the age of thirty-five, but occasionally is seen before the age of twenty-five. The condition is thought to be due to an abnormally high and unregulated concentration of thyroxin in the body brought about by the presence of the adenomatous tissue.

Consequently, the metabolic rate is increased and the clinical manifestations of hyperthyroidism appear. Plummer is of the opinion that in this type of gland the

symptoms are due to the manufacture of an increased amount of normal thyroxin, that the onset is more gradual than in exophthalmic goiter, and that the metabolic rate is lower.

MacCarty has called attention to the fact that areas of hypertrophy are much more common in adenomatous goiters with hyperthyroidism than in adenomatous goiters without hyperthyroidism. Occasionally, microscopic study of an adenomatous goiter with hyperthyroidism does not reveal hypertrophy.

Considerable cardiac injury frequently is associated with this type of goiter. The heart may return to normal, or nearly so, following thyroidectomy which is the accepted treatment for this condition.

Exophthalmic goiter frequently is called Basedow's, Graves' or Parry's disease. It has been suggested that this type of goiter secretes an abnormal product and also an excess of thyroxin. The symptoms produced by exophthalmic goiter are increase in the metabolic rate, a peculiar nervous syndrome, usually exophthalmos, and also a tendency to gastro-intestinal crisis evidenced by vomiting and diarrhea. In exophthalmic goiter there is diffuse enlargement of the thyroid gland.

The use of roentgen rays has been advocated in the treatment of exophthalmic goiter. Many of those who have tried this type of treatment have not been encouraged by the results obtained. Admitting it is efficacious in a limited number of cases, the amount of glandular destruction cannot accurately be measured. Broders has been unable to determine any change in goiters which have previously been treated by roentgen rays. The treatment of exophthalmic goiter is surgical removal. I shall take this up later in this paper.

On microscopic examination, the thyroid glands removed from patients with symptoms of exophthalmic goiter have been shown to present diffuse parenchymatous hypertrophy and hyperplasia in all but a small percentage of cases. In about one-third of the cases, small adenomas may be found which evidently were present before the diffuse parenchymatous hypertrophy took place. The finding therefore of nodular goiters in conjunction with the syndrome of exophthalmic goiter is merely a coincidence.

OPERATION FOR EXOPHTHALMIC GOITER.

In the United States and Canada only forty-five operations for goiter are recorded to 1883. The mortality was slightly less than 15 per cent. Connected with the early surgical work on goiter in this country, the names of Nathan R. Smith, Cooper, Green, Marshall, Fenwick, Halstead, Crile, Ochsner, and C. H. Mayo deserve mention. In Europe, Kocher was one of the first to become interested in the surgical treatment of goiter. At the time of his death, in 1917, about 5,000 patients with goiter had been operated on in his clinic. The surgical mortality of subtotal thyroidectomy for exophthalmic goiter in The Mayo Clinic for the year of 1927 was 0.72 per cent.

Preparation: The improvement in the operative mortality of exophthalmic goiter centers around the efforts that have been made to estimate and reduce the operative risk.

Plummer is of the opinion that an abnormal substance produced in the exopthalmic type of goiter causes the phenomena which characterize the disease; namely, exopthalmos, a psychic state, useless movements, and the gastro-intestinal crisis. He believes that such a patient is always potentially in a condition to be rather suddenly precipitated into an exophthalmic goiter crisis, and that the increased metabolic rate is due to an excessive amount of normal thyroid secretion. Therefore, a marked decrease in the metabolic rate, without a remission of phenomena due to the abnormal secretion, would not indicate that the patient was ready for thyroidectomy. Consequently the operative risk cannot be dependent on the metabolic rate.

Various methods of preparation of patients have been used. In 1921, Pemberton showed that procedures preliminary to surgical procedures, such as ligation and injection into the gland of hot water were used in 123 per cent of the cases. In 1926 and 1927 such preliminary procedures were employed in less than 0.6 per cent of the cases. These methods have been replaced by the preoperative administration of iodin to patients with exophthalmic goiter. This has not only caused decrease in the surgical mortality, but it has greatly reduced the time necessary for surgical preparation and the medical mortality has been practically abolished.

In preparing the average patient with exophthalmic goiter for operation, 30 minims of compound solution of iodin (Lugol's solution) are given daily. The conditon of the average patient may be considered favorable for thyroidectomy in about ten days. This is based on several factors. There is usually marked decrease in the metabolic rate, gain in weight, and marked diminution of the nervousness characteristic of this disease. There may be marked general improvement and only slight decrease in the metabolic rate. The administration of iodin in this type of case stops the progress of exophthalmos and the peculiar nervous syndrome is greatly lessened or abolished.

Usually a crisis in a patient suffering from exophthalmic goiter can be controlled within twenty-four to forty-eight hours. In such a case 50 to 100 minims of compound solution of iodine are given daily, by mouth or by proctoclysis, until the condition is under control. The long continued use of iodin does not permanently relieve the patient with exophthalmic goiter; exacerbations and remissions are still a characteristic feature of the disease.

Technic of Operation.—Since the use of iodin has become established, thyroidectomy has become a comparatively safe surgical procedure. There are, however, some points which should be borne in mind during the surgical removal of goiter. Kocher noticed, in his earlier cases that after thyroidectomy, a peculiar condition occurred which was later recognized as parathyroid tetany. After the discovery of the parathyroid bodies and after their function had been studied, he was able to overcome this common postoperative complication by preserving a portion of the posterior capsule which corresponds with the normal position of the parathyroid bodies.

Another complication, or surgical accident, which must be guarded against is injury to the recurrent laryngeal nerves. The existence of these structures was first discovered and described by Galen, who found that if one of them was severed there was a change in the voice. Formerly it was believed that a fairly common complication following thyroidectomy was collapse of the trachea. It is now known that in practically all of such cases the condition is not that of collapsed trachea but of injury to one or both of the recurrent laryngeal nerves. It is the opinion of many that unilateral and bilateral recurrent laryngeal nerve injury occurs much more frequently than most of us would imagine. A careful survey would lead one to accept this assumption as true. Most injuries to the recurrent laryngeal nerves probably occur because of unfamiliarity with their exact anatomic position, which is quite different from that pictured in the usual textbook. Mikulicz emphasized the importance of leaving a small amount of thyroid tissue on the lateral wall of the trachea, along the course of the nerve to prevent injury to it. Before his suggestion, Billroth reported partial or complete paralysis of the vocal cords in 32 per cent of a series of seventy-one patients operated on by Wölfler. By a statistical study, Jankowski found paralysis of one or both vocal cords in 14 per cent of cases of extirpation of the thyroid gland. The most common sites of injury to the recurrent nerves during thyroidectomy are at the inferior pole of the gland, along the lateral surface of the trachea, and at the

cricothyroid juncture, where the nerve pierces the larynx. If a small amount of glandular tissue is preserved at the inferior pole, injury to the nerve is not likely to occur at this point. If too much of the gland is resected along the lateral surface of the trachea and at the cricoid juncture, and if, also, suturing is necessary to effect hemostasis, the nerve is likely to be injured.

It is extremely important in cases in which general anesthesia is used in addition to local anesthesia, that the patient be allowed to waken after the extirpation of one lobe in order that the operator can determine whether or not there has been injury to the nerve. In most instances injury to one recurrent nerve, of a sort that will be permanent, gives the patient little trouble immediately following operation, and the voice usually approaches normal in two or three months. If severe tracheitis occurs in a case of injury to a single nerve, respiratory embarrassment may be marked for two or three days. If marked stridor occurs, and laryngoscopic examination shows considerable edema of the glottis, with a markedly diminished breathing space, tracheotomy is advisable and should be done at once. Laryngoscopic examination should be made on all patients before thyroidectomy is done. Occasionally, a fixed cord will be found, due to pressure from the goiter. It is wise to make a laryngoscopic examination also immediately following thyroidectomy. In rare instances, symptoms of injury to the recurrent laryngeal nerve develop from two to three days postoperatively. Examination at this time may show fixation of one or both cords whereas immediately following operation their movement may have been normal. In such cases, hot, moist dressings over the wound and inhalations of steam, hasten the subsidence of the condition, which is due to edema or hemorrhage about the nerve. Tracheotomy may be necessary.

Besides injury to the recurrent laryngeal nerves, another complication which may occur is hemorrhage. It is usually manifest from three to twelve hours after operation. The first symptom of this complication usually is respiratory embarrassment. Occasionally, preceding this symptom, the patient may complain of the dressing being too tight. Either of these complaints should receive prompt attention. The dressing should be removed, and if hemorrhage has occurred there will be present marked bulging of the neck. The wound should be opened immediately; if a definite bleeding point can be found and hemostasis effected, the wound may be closed again. If only generalized oozing is found, the wound should be packed with gauze and left open for two or three days, when, under a local anesthetic it may be closed with a resulting satisfactory scar.

SUMMARY.

According to Plummer goiter may be classified as (1) colloid, endemic or simple; (2) adenomatous without hyperthyroidism, or non-toxic; (3) adenomatous with hyperthyroidism, or toxic; (4) exophthalmic, or diffuse parenchymatous, with hyperplasia or hypertrophy.

Diffuse colloid goiter usually occurs at about the age of puberty. It may be largely prevented by the judicious administration of iodin, as shown by Kimball and Marine. Colloid goiter may be successfully treated in young persons by the administration of thyroxin or thyroid extract. This type of goiter is fairly commonly confused with exophthalmic goiter.

Colloid goiters which reach a size sufficient to cause obstructive symptoms should be treated surgically; such cases are comparatively rare. Colloid goiters occasionally contain adenomas which may become hyperactive.

Adenomatous goiter without hyperthyroidism may be classified with colloid or endemic goiter. It apaprently has the same etiology.

The formation of adenomas, except the true tumors, is looked on as a compensatory development in an attempt to meet the demand of the body for thyroxin. In adenomas there may be cystic, calcareous, or hemorrhagic degeneration. If they reach a size sufficient to cause pressure or obstructive symptoms, operation should be employed. The possibility of malignant change is an added indication for surgical removal.

Adenomatous goiters with hyperthyroidism are thought to cause an unusually high concentration in the body of thyroxin, which results in an increase in the basal metabolic rate. This type of goiter may occur before the age of thirty, but usually it occurs after forty-five years of age. The treatment is surgical.

Exophthalmic goiter may occur at any age. It is most common between the ages of twenty and forty. It is thought that there is an excess of normal thyroid secretion in addition to an abnormal thyroid product. The symptoms which characterize it can be diminished or relieved temporarily by the administration of iodin in the form of Lugol's solution. Exophthalmic goiter may be superimposed on adenomatous goiter and rarely on colloid goiter. The ultimate treatment is surgical, the mortality of which has been greatly reduced since the use of iodin has been established.

The most common complications during and following thyroidectomy are injury to the recurrent laryngeal nerves and postoperative hemorrhage.

THE USE OF DRIED BREWERS YEAST IN THE TREATMENT AND PREVENTION OF PELLAGRA.*

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Before the assignment of a specific etiology to pellagra, there was no sound basis for postulating the therapy of the disease. The studies of Goldberger[1] and his associates have, however, gone far to clear up the cause of this illness for they are responsible for the conception that the disease follows a diet containing an insufficient amount of the pellagra preventive vitamin. These investigations stand out as the only sound contribution to our knowledge of the source of pellagra.

The discovery of the use of yeast in the treatment of pellagra came about, like so many other important scientific advances, by accident. In attempting to stimulate the appetite of dogs for a diet calculated to cause them to develop black tongue—a canine disease similar to pellagra in man—Goldberger[2] added yeast cake to their food. Subsequent observation showed that black tongue did not follow a prolonged administration of faulty diet among the dogs to whose food yeast had been added but that an exactly similar ration without the added yeast caused the appearance of symptoms of black tongue in other dogs in due course. Later the removal of yeast from the diet of the first lot of animals was followed by the symptoms of black tongue. These important observations were among the early indications of the role of vitamins in pellagra. It was first though that the lacking vitamin in pellagra was vitamin "B", the anti-beriberi factor, but later it was shown that it was probably a separate entity, which Goldberger designated as vitamin P-P (pellagra pre-

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ventative). It is now, in some quarters, designated as "E". Pecularily enough the search for other foods high in the P-P factor has thus far failed to discover another nutritional element as rich in vitamin P-P as yeast.

PRESENT STUDY.

The increased interest in public health in the flood area of the Mississippi Valley following the spring inundations in 1927 served, among other things, to revive interest in the problem of pellagra. Armed with a new therapeutic agent in the form of yeast, whose usefulness up to that time could be judged only by its action in the laboratory, it was decided to attempt the treatment of pellagra with it in the field.

DESCRIPTION OF STUDY AREA.

Humphreys County has an area of 425 square miles and is located in the flat alluvial delta land of central western Mississippi. The estimated population for 1928 was slightly more than 21,000. In the rural areas the racial ratio is six negroes to one white. County-wide, including towns, the ratio is above 3:1. The chief occupation is agriculture and four-fifths of all agricultural endeavor is connected with the production of long staple cotton. The economic status of the farming population is rated as fair. The diet of both the colored and white farming elements is built around three staple articles—salt-meat, corn-meal and molasses. The most notable recent change in farming practice is that of substitution cash payments monthly to tenants on plantations in place of the former practice of furnishing rations from the plantation commissary. The advent of cheap motor cars has diverted a certain amount of money from the purchase of food and has played a definite part in the increase in pellagra in this part of the south during the past ten years.

OCCURRENCE OF PELLAGRA.

Morbidity — Pellagra is a reportable disease in Mississippi. At the end of each month practicing physicians send to the Health Officer the number of cases, divided as to color, that they have seen during the month. In 1927, 422 cases were reported to the Health Officer during eleven months (there were no reports in the month of April due to the flood), giving a case rate of approximately 21 per 1000 population. During 1928, 547 cases were reported, giving a case rate of approximately 26 per 1000 population. Assuming that 50 cases would have been reported in April, 1927 (a fair average for that month) and counting the influence of free treatment, as will be discussed later, in bringing cases to physicians it is probably still correct to state that there was an increase in case incidence in 1912, but that it was not as marked as the figures would indicate.

Mortality—There were five deaths reported after August 1, 1927, to the Health Department and 16 deaths reported during 1928. Examination of the 1927 death records leads one to believe that all five deaths were due to pellagra (three males and two females under 16 years of age). Closer inspection of the 1928 death record shows five deaths above the age of 65.

Beginning August 1, 1927, the Health Department took steps to popularize the use of dried brewers' yeast in the treatment of all cases of pellagra. A supply of this food was placed in the office of each physician in the county (12 in number) for distribution and a stock kept on hand at the Department. Cases were examined and treated from the Health Department office on written requests from physicians. It soon became the custom to refer all cases to the Health Department and it is on this group that the following conclusions are drawn.

On the first visit, cases were given a superficial physical examination, sufficiently complete, however, to justify the diagnosis of pellagra. The cause of the illness was then explained to the patient and general instructions as to diet were given. The patient was then given 14 ounces of dried brewers' yeast and told to return in two

weeks. Instructions were issued to take one ounce per day (usually divided into three doses each, in a glass of water). When the case returned to the Department at the end of two weeks, another superficial examination was given and a further supply of yeast issued. Thus if patients desired to come in every two weeks during the year, yeast would be given them. Nearly all cases were so benefitted at the end of six weeks that they lost interest and were seen subsequently only on home visits or because of relapses the following spring. During the period reported upon a total of 346 cases were seen; 12 white and 334 colored. Of this group 184 have been seen sufficiently often and studied long enough to draw some conclusions from their cases. One case has already been reported in detail[3] and seven of the remaining are white (three males, four females). In addition 38 other negro cases were kept on a constant dose of brewers' yeast daily for periods varying from 9 to 21 months. This group is discussed separately, later. The reported case and the other white cases are discarded from further study in this report. The remainder of 176 cases, all colored, are divided as to age and sex as follows:

TABLE I.

Age Distribution of Cases—All Colored.

Age	Under 5	5-9	10-14	15-19	20-29	30-39	40-49	50 and over	Total
Male	4	4	5	2	11	5	3	7	41
Female	6	6	4	11	46	29	19	14	135
Total	10	10	9	13	57	34	22	21	176

The above table serves to show three things about this group: First, 75 per cent of the cases occurred in females; second, that over 25 per cent of the cases occurred in females between 20 and 29 years of age and that over 60 per cent of all cases occurred in females above 20.

SYMPTOMS AT ORIGINAL EXAMINATION.

As each case reported to the Health Department for treatment, note was made of the outstanding symptoms of which the patient complained. Following is a table showing the results of this inquiry.

TABLE II.

Showing Complaints on First Examination.

Mouth symptoms only	19
Skin symptoms only	49
Mouth and skin symptoms	108
Diarrhea	34
Constipation	19
No complaints referable to bowels	123
Other symptoms (nervous)	3

This table shows that, when first seen, 69 per cent of the patients conplained of mouth and skin symptoms, while 28 per cent of the group complained of skin symptoms only. Seventy per cent of the group had no complaint referable to the bowels and only 17 per cent complained of diarrhea. The last statement is interesting in that, as described in earlier studies, diarrhea was considered one of the cardinal symptoms of pellagra.

Patients were questioned at the first examination in regard to previous pellagra. The results of this interrogation were as follows:

New cases 95, first relapse 56, second relapse 10, third relapse 5, fourth relapse 4, fifth relapse 2, sixth relapse 2, eighth relapse 1, tenth relapse 1.

TREATMENT.

The total amount of yeast issued to the group amounted to 527 pounds (8,432 ounces), on an average of slightly over 48 ounces per case. It may be assumed that the average duration of treatment of these cases was about 50 days.

From the very nature of such therapy it will be seen that the use of brewer's yeast is a temporary measure; its function is to place in the diet a lacking element and if the yeast is withdrawn and the lacking vitamin is not obtained from other sources there is no reason to expect anything but a relapse of the disease in the near future. Such a therapy must be regarded, then, in

the light of a palliative measure to be used in tiding patients over an acute attack of the disease and carrying them along until their diet can be so extended as to prevent the recurrence of symptoms.

Of the 176 cases reported here as treated during at least one attack of pellagra, 175 were alive on April 1, 1929; one case died during treatment after five weeks. All other cases were, according to their statements and according to our examinations, benefitted by the use of yeast. The usual course of recovery begins with an improvement in the dermatitis, followed rather soon by a disappearance of soreness in the mouth. At the end of two weeks the dermatitis has almost disappeared and the subjective mouth symptoms are gone. At the end of four weeks there are usually no complaints, but the mouth and tongue pathology, although improved, is still obvious. The glossitis seems to be the last symptom to disappear.

The exact value of this treament is difficult to estimate, however, when it is remembered that the ordinary case of pellagra that does not terminate fatally is usually clear of symptoms by October of the year in which symptoms develop.

One hundred and four had suffered no relapses in a period averaging 11 months after completing an average of 50 days of treatment. This group represents those cases who, having cleared up from the attack for which they sought treatment, have not returned for further aid. Information concerning them was obtained by home visits. Twenty-three cases that had likewise stopped treatment, when visited in February, 1929, showed early symptoms of recurrence (sore mouth and gastro-intestinal complaints). A group of 38 cases, as a special study, were kept on daily doses of one ounce of yeast from May until October and one-half ounce daily from October to May. The oldest case has been kept 21 months without relapse. In this entire group, averaging 13 months of treatment according to the schedule above, there has been no relapse.

RELAPSE.

As noted above, 23 treated cases showed symptoms of relapse later when found by home visits. Of the cases voluntarily returning to the office for further treatment, 10 definitely showed symptoms of relapse. It is noted in passing that these 10 cases had averaged only 19 days of treatment.

SUMMARY.

Of 176 cases studied, one died during treatment, 104 had suffered no relapses in 11 months after 50 days treatment, 33 cases showed symptoms of relapse within one year after slightly under 50 days of treatment, and 38 special cases kept on daily doses averaging about three-fourths of an ounce the year round showed no relapses after an average of 13 months' observation.

CONCLUSIONS.

1. The results of the treatment of 176 cases of pellagra, with dried brewers' yeast, in negroes in Humphreys County, Mississippi, since August 1, 1927, are given.

2. Dried brewers' yeast in doses of one ounce per day greatly assists in allaying all symptoms of pellagra.

3. Once arrested, unless a dietary change is instituted to supply the missing vitamin, the withdrawal of yeast is followed by relapses within a year in about one-third of the cases.

4. A daily dose of one ounce of dried brewers' yeast during the period, May to October, and a half an ounce daily from October to May prevented the recurrence of symptoms of pellagra in all of 38 cases, over a period averaging 13 months.

5. It is pointed out that the ultimate control of pellagra is a matter of education in dietetics and that the use of brewer's yeast should be confined to the actual treatment of cases and to use for prophylactic reasons only until cases may be gotten on a diet that in itself will prevent the disease.

6. The subject invites further study.

BIBLIOGRAPHY.

(1) Goldberger, Joseph; Wheeler, G. A.; Sydenstriker, E., and King, W. I.: Study of endemic pellagra in some cotton mill villages of South Carolina. Pub. Health Rep., 43:2645-2647, 1928. And enormous other publications by Goldberger in Public Health Reports.

(2) Personal Communication at Hot Springs, Arkansas, Sept. 17, 1928.

(3) Carley, Paul S.: Case of pellagra following voluntary reduction of diet, J. A. M. A. 91:879-880, 1928.

An excellent short history of the use of yeast in medicine together with a bibliography of fifty-six references on the yeasts is to be found in an article by C. Faust-Newton, "The Uses and Therepeutic Value of Yeast and More Particularly of Dried Brewers' Yeast," M. J. and Record 123:689-691, 1926.

DISCUSSION.

Dr. R. D. Dedwylder (Cleveland): Mr. Chairman, Ladies and Gentleman: Dr. Carley has so thoroughly covered this subject that it leaves very little for me to say other than to back up what Dr. Carley has said in his remarks on the use of brewer's yeast in the treatment of pellagra.

All my thoughts are based on the observation of several hundred cases of pellagra treated following the Mississippi Valley flood in 1927. The Red Cross supplied us with all the yeast we needed. In that way, my observation did cover treating several hundred people suffering more or less from pellagra.

To my mind, it is inconceivable how a man on a well-balanced ration can develop pellagra. You can readily see then that I think pellagra is a nutritional disease, and to my way of thinking, nutrition and nutritional disturbances are one of the biggest factors with which we have to contend, especially in the tenant class of people in the Mississippi Delta Valley.

Brewer's yeast furnishes us with a chief source of a concentrated pellagra preventive principle that we can use as a food in the treatment of pellagra. It will keep the individual going and relieve him of all symptoms until you can supply the pellagra preventive principles in some other forms, if you are able to do it at all.

There is one thing relative to nutritional disturbances of pellagra that I do want to mention other than what Dr. Carley has brought out. A good many of the laymen feel that pellagra is a food sensitive disease, that it is brought around from eating impure maize or from eating maize of any kind, corn meal, corn bread, etc.

That is not so. It is due to an unbalanced ration. If you get them off one side and put them on the other extreme, you still have these people on an unbalanced ration and you will have other disturbances. In the management of pellagra you want to get the individual on a well balanced ration. That is the secret of the whole business. As long as the patient is maintained on a balanced ration, he will not have pellagra or these nutritional disturbances.

By way of parenthesis, I suspect there are some dog lovers, bird hunters and fox hunters here. Yeast will relieve dogs of black tongue, and if dogs are fed yeast it will prevent them from developing black tongue. We have done this in several instances in our work in that county from an experimental standpoint to see if it would bring out these facts.

Further than these matters I have stated, I have nothing to add to the paper. The main thing that I wish to leave is that it is not a food sensitive disease, but a food deficiency disease.

Dr. Paul S. Carley (Closing): I have very little to add with the exception of one or two remarks. One of the last communications I had from Dr. Joseph Goldberger before he died impressed me very distinctly of trying to impress people generally that brewer's yeast is not a prophylactic against pellagra. I had a case of pellagra which was treated with yeast and was cured of symptoms, but when withdrawn from the yeast, the pellagra reappeared.

There is a widespread use of neo-arsphenamin in the treatment of pellagra. However, there are excellent physicians in my county who use yeast who formerly relied on the use of neo-salvarsan.

We have been studying the matter from the incidence of syphilis in the general population. The figures quoted by Van Alstine are used as a base line for syphilis in the general population. We have found to date in about two hundred cases which we have examined that the syphilis rate is lower in pellagra than in the general population rate. Whether or not that will hold out later, I do not know.

From the standpoint of syphilis, according to our own figures, we cannot determine that the syphilitic rate in negroes is as high as it is in the base line of the general population, which I think is somewhat fallacious.

SYMPOSIUM ON INFECTIONS OF THE LIVER AND SUBPHRENIC SPACE. MEDICAL, ROENTGENOLOGICAL AND SURGICAL ASPECTS.

I. ETIOLOGY AND DIAGNOSIS OF SUPPURATION IN AND ABOUT THE LIVER.*

J. H. MUSSER, M. D.,†

NEW ORLEANS

One of the very real problems of medical practice is the detection of foci of suppuration either in the liver or in the immediate neighborhood of this large organ. Because of the relation the hepatic tissue has to the diaphragm, so-called subphrenic abscesses are frequently likely to occur in this region. This, then, is the first and the most important type of suppurative lesion that occurs about the liver. It might be mentioned in passing that in this symposium I will omit completely any reference to infections of the gall-bladder or bile tracts. The second important cause of suppurative lesions, this time within the liver, is a portal pylephlebitis, which is responsible for multiple abscesses of the organ. The third and by far the most frequent cause of liver abscess is the almost ubiquitous Endameba dysenteriae, which is responsible for large single abscesses. In so far as the multiple abscess is concerned the usual causes are those dependent upon an intra-abdominal inflammatory disorder. Multiple abscess of the liver may be due to a host of causes and these causes consist of any factor that would produce multiple abscess in any part of the anatomy, but for some special reason seems to occur more frequently secondary to acute osteomyelitis. In this condition, however, the picture is one of a general process, a septicopyemia, rather than of local infection of the organ under discussion.

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†(From the Department of Medicine, Tulane University School of Medicine).

SUBPHRENIC ABSCESS

I will discuss first subphrenic abscess. The accompanying table will explain in detail the etiologic factors responsible for subphrenic abscess in 84 cases as reported by Fifield and Love.(1) From this chart it may be noted that appendicitis is a most frequent cause; but if the various gastric etiologic factors were grouped, they would outnumber the cases of appendicitis responsible for this condition. Ochsner(2) states that the most frequently involved subphrenic space is the right posterior superior and next, the right inferior.

TABLE I

ORIGIN IN 84 CASES OF SUBPHRENIC ABSCESS COLLECTED BY FIFIELD AND LOVE

	No. of Cases	Per cent of all cases
Appendix	30	35.7
Perforated Duodenal Ulcer	12	14.2
Perforated Gastric Ulcer	12	14.2
Operations on Stomach	9	10.7
Gall-Bladder	5	5.9
Kidney	2	2.3
Chronic Gastric Ulcer	1	1.1
Carcinoma of Stomach	1	1.1
Carcinoma of Esophagus	1	1.1
Fractured Pelvis	1	1.1
Rib	1	1.1
Unknown	9	10.7

SYMPTOMS.

Non-suppurative subphrenic infections are probably much more common than they are thought to be. They may occur with fairly definite signs and subside spontaneously. The constitutional symptoms of subphrenic abscess are those common to any infection or to a localized collection of pus from which there is a certain amount of absorption taking place. Thus the fever, the leukocytosis, and constitutional reaction in general indicate that there is infection present somewhere in the body. Localized symptoms early in the course of the infection are usually not outstanding. It seems safe to assume, however, in an individual who has had an acute suppurative appendi-

(1) Fifield, L. R. and Love, R. J.: Subphrenic abscess, Brit. Jour. Surg., 13:683, 1926.

(2) Ochsner, A.: Subphrenic abscess, New Orleans Med. and Surg. Jour., 81:102, 1928.

citis or a perforated peptic ulcer that the subphrenic space may be involved. Steps may be taken to prove this, consisting of a demonstration of localized tenderness, usually over the tip of the twelfth rib. Occasionally there is pain in the lumber region and some tenderness there. The pleural physical signs are of interest often because of the reaction of the diaphragm. Littlen's sign, the shadow of the diaphragm moving during respiration, is very much less pronounced on the affected side than on the other side. Limitation of the movement of the diaphragm can be demonstrated by percussion, marking on the skin with a dermatographic pencil the upper and lower limits of pulmonary resonance on deep inspiration and expiration, which is normally on the left side about a centimeter greater than on the right. If there is an extension upwards through the diaphragm of the inflammatory products, an acute pleurisy develops. This tends still further to immobilize the diaphragm. The pleurisy may be of the plastic type or may be primarily followed by the outpouring of fluid, usually in relatively small amounts. Under any circumstances, whether plastic or fluid, there will be further dullness on percussion, diminished tactile fremitus, vocal resonance and distant sounds, but with maintenance of normal respiratory rhythm. Later in the course of the condition there may be marked elevation of the diaphragm, which can be confirmed by the high level of liver posteriorly, or superiorly in a certain proportion of cases.

Diagnosis of subphrenic abscess is evidently extremely difficult. This would explain the high mortality rate of the condition because were the surgeon gifted with eyes on the end of his fingers, he might be able to localize the area of suppuration and if this were done relatively early, he would not be obliged to operate upon a patient worn out and worn down by a long continued illness.

PYLEPHLEBITIS.

Pylephlebitis occurs in the great majority of cases secondary to acute suppurative lesions in and around the appendix. It is responsible for some 0.1 of 1 per cent to 5 per cent of patients dying of acute appendicitis. It is the most common cause of he-

TABLE II

INCIDENCE OF PYLEPHLEBITIS IN ACUTE APPENDICITIS[1].

Author	Cases of Acute Appendicitis	Cases of Pylephlebitis	Per Cent of cases
Stillmann	545	2	.14
Hoffman	4,000	7	.17
Moschcowitz	1,529	7	.45
Brutt	2,500	15	.61
Gerster	1,187	9	.76
Quertz	533	4	.75
Clairmont and Meyer	1.187	4	.33
Fitz	257	11	.4
	(Suppurative)		
Gibson	782	1	.13
Braun	600	8	1.3
Colp	2,841	9	.3
Reock	147	2	1.3
Bell	1,726	8	.46
Petren	170*	14	8.2
Armstrong	546*	28	5.1
Langdon-Brown	9,494*	12	.12

*Autopsy Cases.

patic abscess in non-tropical countries. In addition to portal pyleph'ebitis, it is, of course, possible to have ineffective emboli carried from the appendiceal veins to the liver without involvement of the vein itself. This, however, seems to be rare.

SYPMTOMS.

Again given the patient who has had an acute appendicitis, the possibility of acute pylephlebitis is considered when the patient has a markedly rapid rise in temperature accompanied by severe chills and an increase of the local symptoms. The marked constitutional reaction is indicative of septic material entering the portal circulation, possibly the general circulation. These severe chills seem to be the most characteristic features of tht disease. Some authors go so far as to say that the severe chill is a pathognomonic sign of portal empyema when there is an inective pro-

cess in the area drained by the portal vein. Chills are, of course, followed by fever and profuse sweats. There is frequently pain in the liver region and the liver is enlarged and tender. As a resu't of the suppurative hepatitis the enlargement of the liver may be so great as to push the diaphragm upward and give to an exaggerated degree the diaphragmatic phenomena just mentioned in conjunction with subphrenic abscess.

The condition of pylephlebitis is a difficult one to relieve. The most important factor is early and prompt operation of the suppurative appendix when marked fluctuation in the temperature, severe chills, and high leukocytosis are present.

AMEBIC ABSCESS.

The third type of liver infection, the amebic abscess, is the one which is more familiar to the clinician and internist than are the other two, certainly, at least, in our section of the country. As a matter of fact, the great majority of the abscesses of the liver are amebic, but it does not necessarily follow that because a man has amebic dysentery that he is going to develop an amebic abscess. Statistical studies have been made of dysentery of the amebic type in the tropics and elsewhere and as do statistics so often, these figures vary considerably. Simon (Tice's Practice) says that hepatic abscess occurs in at least 20 per cent of the fatal cases of amebic dysentery. These figures, however, are very much higher than is the actual instance of the condition, because there are a large number of carriers of Entameba histolytica who rarely develop symptoms of any kind, and there are a considerable number of patients who never have very much more than the slight diarrhea. In approximately one hundred cases that I have seen, a liver abscess occcurred in a little over 2 per cent of these cases.

The first liver abscess that I saw was in Philadelphia. A tentative and presumptive diagnosis was made of gumma of the liver and the patient was given active antisyphilitic treatment. Only after failure of the active treatment to eradicate the presumed syphilis, did the thought occur that the condition might be an abscess.

Hepatic abscess of the liver may be divided into two types, and utilizing the classic description of aneurysm, the abscess may be one of signs or one of symptoms. However, it is impossible to draw a clear cut line between the two. As may be inferred, the abscess of signs is one of comparatively little constitutional reaction on the part of the patient. The liver is enlarged on palpation and by percussion, and it may be that the abscess will actually point towards the anterior abdominal wall.

The abscess of symptoms is the one in which we have the most typical picture. The patient has fever, sometimes considerably elevated, chills and night sweats. Pain is frequently present and this pain because of the involvement of the diaphragm may be referred to the neck or shoulder region. Whi'e the shoulder pain is spoken of as *the* referred pain of liver abscess, it would seem that neck pain is somewhat more common. With this neck pain there is associated skin hyperalgesia. This type of abscess is the one in which the acute hepatitis has gone on rapidly to suppuration. The abscess of signs is usually the one which has developed slowly, insidiously, and has a thick capsule so that systemic absorption is reduced to a minimum.

The amebic abscess, it is true, is usually single, but there is no reason to feel definitely and positively that this is going to be the case, because multiple abscesses are apparently reasonably common. Approximately seven out of ten abscesses occur in the right lobe. The only explanation for this is that the right lobe is considerably larger; also it is supposed that that the intestinal blood goes largely to the right

lobe of the liver. The right lobe abscesses have a tendency to point upward toward the diaphragm and if there is rupture from the diaphragm into the lung, many complications may occur, even a hepato-, bilio-, tracheal fistula with bile appearing in the sputum. This is rare.

Frequently the abscess may be a combination of the two types mentioned above. It is this type that so frequently gives the very misleading physical signs of pleurisy. The abscess comes in close contact with the diaphragm, infected material is carried by way of the lymphatics through the pleura and a pleural reaction occurs. This may occasion a frank pleural effusion or more than likely it may occur that there is no outpouring of fluid but merely a proliferative response to injury. Under any circumstances, both conditions will cause dullness on percussion or at least impairment of percussion, a diaphragm which is immobile or movement is very limited. This particular physical sign is sometimes hard to demonstrate because of the inability to differentiate between liver dullness and pleural dullness. The same statement applies to the frequently mentioned irregular line of dullness of the upper border of the liver posteriorly. Naturally with the impairment of percussion, breath sounds are found to be weakened and tactile fremitus is diminished. Frequently the vocal resonance is least impaired.

It seems hardly necessary to reiterate the oft-made statement that abscess should be diagnosed before rupture through the diaphragm or before bulging and fluctuation take place. This may be true, but nevertheless, there is frequently considerable difficulty in arriving at a diagnosis, particularly if the patient is not seen relatively early in the disease, when there is an absence of leukocytosis, and when there are no amebas in the stools.

SUMMARY.

There are three important etiologic factors responsible for suppuration in or about the liver, excluding infection of the bile passages. A hepatic amebic abscess is the most common, followed in order by subphrenic abscess and portal pylephlebitis. As the infection is usually in more or less close relation to the diaphragm, physical signs, including here the ordinary diaphragmatic phenomena, may be more definate in the thorax than in the abdomen.

II. RADIOLOGICAL SIGNS OF SUB DIAPHRAGATIC ABSCESS*.

AMEDEE GRANGER, M. D.,†

NEW ORLEANS

The principal radiological sign of subdiaphragmatic abscess is an abnormal elevation of the diaphragm, and as this must of necessity cause dullness over the lower region of the thorax it is very easy to understand why, given an incomplete or unreliable history, so many of these cases are sent to the radiologist with a clinical diagnosis of unresolved pneumonia, empyema or pleural effusion.

Two good radiographs, one a P. A. and the other a lateral view, preferably made at a target-plate distance of six feet and with the patient standing or sitting, or a thorough fluoroscopic study will show with certainty that the pathological process is above or below the diaphragm; and in the rarer cases with pathology both above and below the diaphragm this will be revealed also.

The radiological examination having established the fact that the pathology is subdiaphragmatic, let me remind you that this examination forms an integral part of the whole diagnostic procedure, and that although at times the most important part, it is comparatively rare that a correct diagnosis can be made from the radiological examination alone, without the

*Read before the Orleans Parish Medical Society, November 11, 1929.

†From the Department of Radiology, Graduate School of Medicine, Tulane University and Department of Radiology, Charity Hospital.

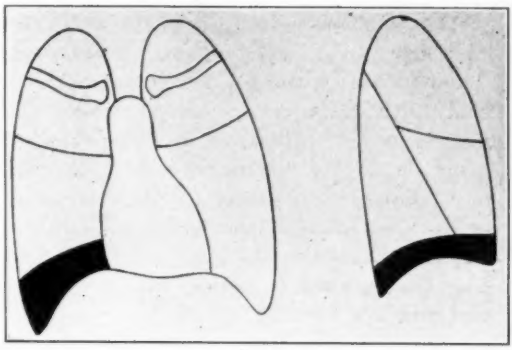

Fig.I. Schematic reproduction of a radiograph of a sub-diaphragmatic abscess resulting from a liver abscess.

knowledge of certain correct and essential clinical data. And these should be obtained and corelated with the radiographical findings before the latter are interpreted in terms of pathology and conclusions are drawn.

In our experience the most frequent cause of sub-diaphragmatic abscess was abcess of the liver, but it is necessary to consider such causes, as visceral perforations, perinephritic abscess, infected large hematoma resulting from contusion of the back and empyema necessitates pointing through the diaphragm, and to exclude them before making a final diagnosis of sub-diaphragmatic abscess, resulting from liver abscess.

This has not proved difficult when a careful physical examination was made and a pains-taking history was obtained.

I desire to particularly call your attention to a type of elevation of the diaphragm which I have never seen except when the sub-diaphragmatic abscess was due to a liver abscess. Please note (Figure 1) in the schematic reproduction of a typical radiograph of a case of sub-diaphragmatic abscess resulting from liver abscess, besides the elevation of the diaphragm, the obliteration of the cardio-phrenic angle in the P. A. view and of the anterior costo-phrenic angle in the lateral view.

Our experience at the Charity Hospital since first I noticed this sign about six years ago, would seem to justify my belief that it is diagnostic of sub-diaphragmatic abscess secondary to liver abscess.

The next schematic reproduction (Figure 2) is of a typical radiograph of a sub-diaphragmatic abscess arising as a result of a more or less general peritonitis from any of the causes previously enumerated including abscess of the liver. Please note that in this instance it is the costo-phrenic (instead of the cardio-phrenic) angle which is obliterated in the P. A. view and the posterior costo-phrenic (instead of the anterior costo-phrenic) angle which is obliterated in the lateral view.

In cases of liver abscess without sub-diaphragmatic abscess the diaphragm was found elevated with the costo-phrenic and cardio-phrenic angles diminished, but not obliterated.

And in still other cases of liver abscess without sub-diaphragmatic abscess a small but distinct bulging or pointing upwards into the lower lung field (Figure 3) was seen. A gumma of the liver could produce an identical picture, but a fluoroscopic examination would show that the movement of the diaphragm was not restricted to nearly the same degree that it would be if the case was one of liver abscess.

When there existed a distinct reaction in the pleura or in the lung surrounding the localized bulging of the diaphragm and liver it was predicted correctly that rupture into

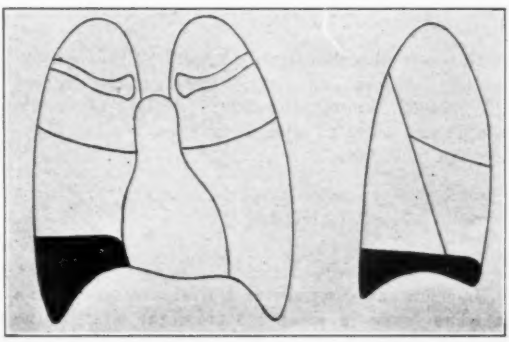

Fig. II. Schematic reproduction of a radiograph of a sub-diaphragmatic abscess resulting from various causes including liver abscess.

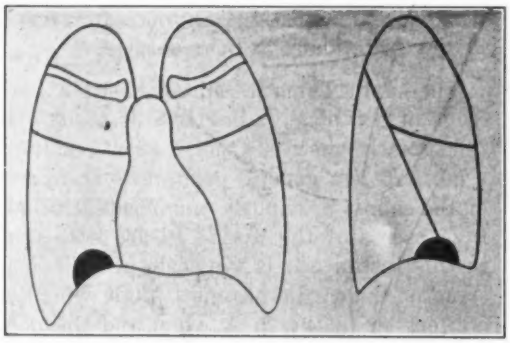

Fig. III. Schematic reproduction of a radiograph of a liver abscess pointing upward.

the lung would take place within 24 to 72 hours.

We believe with Pancoast that the apparent lobar pneumonias in cases of liver abscess (Figure 4) are not true lobar pneumonias, but exaggerated lung reactions due to the proximity of the abscess to the upper surface of the liver. The temperature charts are not those of a lobar pneumonia. We known that extensive areas of congestion and edema can surround any large abcess and that edema of the skin and subcutaneous tissue may overlie a liver abscess. It seems reasonable that a similar reaction could arise in lung tissue. Moreover a moderate amount of clear fluid is frequently found in the pleural cavity. Therefore, we have adopted the suggestion of Dr. Eliason and call these lung reactions a pulmonitis.

ILLUSTRATIVE CASES

1. Abscess of the liver.

G. G.—White male, aged 34 years. Clinical diagnosis: Abscess of liver. Roentgen-ray report No. 124363, August 10, 1929: Right leaf of the diaphragm elevated with diminution but not obliteration of its angles.

The most probable cause is a hepatitis with or without abscess formation.

Operation: 8-13-29.

Incision and drainage of a liver abscess. Motile amebae found in scrapings from the wall of the abscess cavity.

2. Abscess of the liver, on point of rupturing into the lung.

F. H.—Colored male, aged 33 years. Clinical diagnosis: Right pleural effusion. Roentgen-ray report No. 115417, March 4, 1929: No evidence of fluid in the pleural cavity. The abnormally high position of the right leaf of the diaphragm with wooly outline, the definite evidence of inflammatory reaction of the pleura and the enlargement of the lower peri-bronchial lymphatics would all seem to indicate that the process is caused by a liver abscess, which is probably near the point of rupturing into the lung.

In the afternoon of March 5, Dr. Mattingly reported that the patient was expectorating what looked like liver pus.

On March 6, Dr. Jamison aspirated 50 c.c. of typical anchovy sauce fluid from the liver.

Later that day he was operated on under ether anesthesia and about 1½ pints of pus evacuated from his liver.

3. Abscess of the liver—pointing.

V. B.—White male, aged 48 years. Clinical diagnosis: Gastric carcinoma. Probable liver abscess. Roentgen-ray report No. 108025, Oct. 8, 1928: G. I. series showed no evidence of organic pathology of the stomach and bulb. The stomach is seen entirely to the left of the spine. The right leaf of the diaphragm is elevated, and fluoroscopic study showed that its movement was markedly limited. Roentgen-ray report No. 109422, Nov. 3, 1928: Localized bulging (cone shape) in the posterior half of the right leaf of the diaphragm, with adhesions between the diaphragmatic and visceral pleurae. These signs would indicate a liver abscess pointing upward.

On Nov. 22, 1928, the liver was aspirated and pus found.

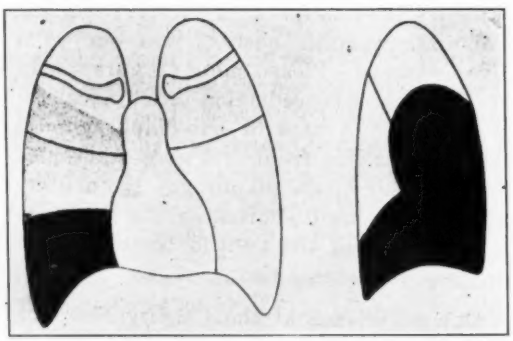

Fig. IV. Schematic reproduction of a radiograph of a sub-diaphragmatic abscess resulting from a liver abscess and complicated with a pulmonitis of the lung.

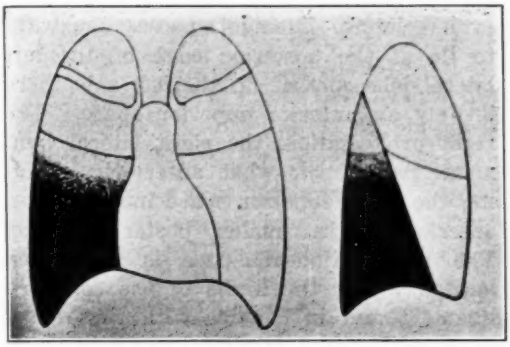

Fig. V. Schematic reproduction of a radiograph of a lobar pneumonia of the lower lobe of the right lung.

4. Sub-diaphragmatic abscess from abscess of the liver.

E. S.—White female, aged 20 years. Clinical diagnosis: Sub-diphragmatic abscess. Roentgen-ray examination, March 19, 1929, showed a marked elevation of the diaphragm with complete obliteration of the anterior costophrenic angle and a localized pulmonitis of the anterior region of the lower portion of the right lung. This clearly indicated a sub-diaphragmatic abscess due to liver abscess with secondary reaction if not actual infection of the base of the right lung.

March 27 the patient began expectorating some thick brownish purulent material.

5. Sub-diaphragmatic abscess from liver abscess.

N. C.—White male, aged 30 years. Clinical diagnosis: Liver abscess. Roentgen-ray report, No. 124009, Aug. 1, 1929: The marked elevation of the right leaf of the diaphragm, with clear lung field above it, and the marked diminution of the costo-diaphragmatic angles in the axillary zone of the P A view and the posterior zone of the lateral view indicate clearly a sub-diaphragmatic collection of fluid or pus. Careful history should determine whether this process results from a liver abscess or some other cause.

At operation Aug. 3, 1929, a large quantity of very thick yellow pus was evacuated, and digital exploration revealed an irregular cavity which was unquestionably in the liver.

6 Sub-diaphragmatic abscess from abscess of the liver complicated by a pulmonitis of the right lower lobe.

V. M.—White male, aged 40 years. Clinical diagnosis: Abscess of the liver. Roentgen-ray report No. 119834, Nov. 18, 1929: Right diaphragm markedly elevated and consolidation of the lower lobe of the right lung from a pulmonitis, most probably due to extension of infection or rupture of a sub-diaphragmatic liver abscess.

Operation on May 26, 1929 when a large amount of pus was evacuated from a liver abscess.

Roentgen-ray examination on June 6, 1929, revealed the right diaphragm much lower than at the last examination and the pulmonitis cleared up except for a small area of consolidation in the lower and posterior portion of the right lower lobe.

7. Abscess of liver—later with sub-diaphragmatic abscess.

A. D.—White male, aged 8 years. Clinical diagnosis; empyema pleurisy. Roentgen-ray report No. 127063, Sept. 30, 1929: There is no evidence of pathology in the thorax. The right diaphragm is elevated and its angles are diminished. The most probable cause is abscess of the liver.

A few days later an abscess in the liver was opened and drained. As the patient did not do as well as was expected he was re-examined on Nov. 26, 1926, when roentgen-ray report No. 128389 read—The increased elevation of the right diaphragm with obliteration of tht cardio-phrenic angle would indicate a sub-diaphragmatic abscess arising from liver abscess.

At operation a day or so later this diagnosis was confirmed and the abscess drained.

CONCLUSIONS.

Abnormal elevation of the diaphragm is the principal and most constant radiological sign of sub-diaphragmatic pathology.

Elevation of the diaphragm with obliteration of the cardio-phrenic angle in the P. A. view and of the anterior costo-phrenic angle in the lateral view means a sub-diaphragmatic abscess secondary to a liver abscess.

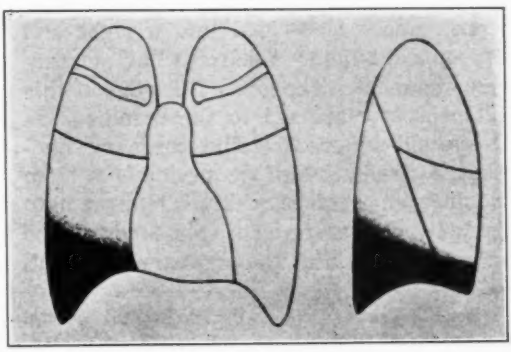

Fig. VI. Schematic reproduction of a radiograph of an empyema.

III. SURGICAL TREATMENT OF SUBPHRENIC INFECTIONS.*

ALTON OCHSNER, M. D.†

NEW ORLEANS.

Lesions within the subphrenic space have been of considerable interest to surgeons for a long period of time. Because of the relative inaccessible location, protected by the thoratic cage, even though lying within the abdominal cavity, these processes immediately below the diaphragm have been difficult to diagnose and also to treat. Of surgical interest subdiaphragmatic infections are most important, first, because these inflammatory lesions follow a suppurative process within the abdominal cavity, and, second, because the treatment of the condition is entirely surgical, even though the majority of cases require no operation.

The subphrenic space may be designated as that space located between the diaphragm above and the transverse colon and transverse mesocolon below[1]. The inclusion of the area beneath the liver and between it and the transverse colon and transverse mesocolon is desirable, from a surgical standpoint, because not infrequently both supra- and infra-hepatic inflammatory lesions occur concomitantly. A knowledge of the anatomical spaces in the subphrenic area is essential, as the surgical treatment, including the operative approach, is dependent upon the location of a subdiaphragmatic infection. The supra-hepatic space is divided into a right and left space by the falciform, or suspensory, ligament. The lower free edge of this ligament is continued to the umbilicus as the round ligament. The coronary ligament is a reflection of the peritoneum from the inferior surface of the diaphragm to the superior surface of the liver. The left prolongation of the coronary ligament, or left lateral ligament, passes backward to lie at the posterior edge of the left lateral lobe so that on the left side there is only an anterior superior space. The right prolongation, the right lateral ligament, passes somewhat anteriorly, dividing the right superior space into a larger anterior and a smaller posterior space. The retroperitoneal space is that area located within the limits of the coronary ligament and in contact with those portions of the liver and diaphragm which are not covered by peritoneum. The infra-hepatic space, which is located between the liver above and the transverse colon below, is divided into a right and left inferior space by the round ligament and the ligament of the ductus venosus. The left inferior space is again divided into an anterior and a posterior space by the lesser omentum, the space located posterior to the lesser omentum, being the lesser peritoneal cavity, and that anterior being the left anterior inferior space.

The "right posterior superior" space is the most frequent site of an inflammatory focus in the subphrenic area. In a series of 78 cases of subphrenic abscess reported by Fifie'd and Love[2] from the London Hospital the "right posterior superior" space was involved in 20 instances, an incidence of 37.1 per cent. An abscess in the "right posterior superior space" is frequently complicated by a similar suppurative process beneath the liver in the "right inferior" space. The extra peritoneal space, formed by the coronary ligament on the superior surface of the liver, was the site of an abscess formation in 20 of Fifield and Love's[2] cases, (25.6 per cent).

The etiology of subphrenic infection has been discussed by Dr. Musser, but, even at the risk of repetition, I wish again to emphasize the fact that the most frequent etiological agent in subphrenic abscess is acute appendicitis, the incidence of this complication in acute appendicitis varying from .3 per cent to 4.9 per cent (Bancroft[3], Stillman[4], Beekman[5], Clair-

*Read before the Orleans Parish Medical Society, November 11, 1929.

†From the Department of Surgery, School of Medicine, Tulane University, New Orleans. La.

mont and Meyer[6], Cutler[7], and Suermondt[8]). Most cases of subphrenic infection subside spontaneously without suppuration, so that inflammatory processes occur in the subphrenic space much more frequently than statistics, based on the finding of localized abscesses, indicate. In a group of 972 cases of acute appendicitis Neuhof[9] reported 15 such cases of subphrenic infection which did not progress to suppuration. Similar cases have been reported by Lee[10], Clendening[11], and the author[12]. The treatment of such an infection is non-operative, and consists of rest, immobilization of the affected part, as well as the homolateral hemithorax, by means of adhesive plaster, and the application of heat locally. Early in the condition it is impossible to predict whether suppuration or absorption will ensue. Not infrequently subphrenic, non-supprative inflammatory processes are not recognized clinically by the attending physician, because of the relatively few signs and symptoms presented, and, as has been emphasized by Dr. Musser, the diagnosis of a frank subdiaphragmatic suppuration is often delayed for a considerable period of time.

The prognosis in subphrenic abscess is bad. The mortality in the reported cases varies from 23 to 100 per cent (Douglas[13]—33 per cent; Hodges[14]—50 per cent; Eicher and Kibzey[15]—50 per cent; Bauman[16]—66 per cent; McEachern[17]: operated cases—33 per cent, non-operated —75 per cent; Tuft[18]—66 per cent; Lotsch[19]: operated cases—33 per cent, unoperated cases—100 per cent.) Fifield and Love[2] report a general mortality of 50 per cent in their cases. Of 59 cases operated upon there was a mortality of 32 per cent. Lockwood[20] states that in unoperated cases there is a mortality of from 85-100 per cent, whereas in the cases subjected to operation there is a mortality of 66 per cent. The author[12], in a previous communication, reported 15 cases with only one fatality, a mortality of 6.6 per cent.

The mortality in subphrenic abscess has, in the past, been considerably higher than in residual abscesses located elsewhere in the peritoneal cavity. This higher death rate in subphrenic abscesses is probably due to several factors: first, because of the relatively inaccessible location of the abscess, a diagnosis is made late, i. e. not until after a toxemia has become well established; second, because of the intimate relation of the inflammatory process to two large serous cavities, the possibility of an infection occurring in either one or both of these cavities, as the result of rupture or extension, is great. This applies especially to the pleural cavity, as an infection of the pleural cavity occurs invariably in cases of subphrenic abscess which have existed a long time. The high mortality following surgical drainage of subphrenic abscess has in the past probably been due to contamination and subsequent infection of a previously uninvolved serous cavity. Even though empyema or peritonitis in themselves may not cause the death of the individual, an infection of either one of these large serous cavities, in addition to the loca'ized suppurative process in the subphrenic area, may produce death.

The surgical approach to a subphrenic abscess offers certain technical difficulties because of the location of the process. The most frequently employed routes of drainage have been, in the past, transpleural or transperitoneal. The drainage of any infected area through a non-infected serous cavity violates all surgical principles. The drainage of a subphrenic pyogenic abscess through an uninvolved pleural or peritoneal cavity is as unsurgical as drainage of a pulmonary abscess through an uninvolved pleural cavity. Fifield and Love[2] report a mortality of 47.3 per cent in those cases of subphrenic abscess which were drained transpleurally and a mortality of 23.8 per cent in those cases which were drained through the peritoneal cavity. In order to prevent an infection of either the pleural or peritoneal cavity, it is impera-

tive to drain a subphrenic abscess without entering either of these cavities, or at least without entering an uninvolved portion of either serous cavity. This can be readily accomplished in the majority of cases. An abscess located in the "right posterior supererior space" (which is the most frequent site of a subphrenic abscess) may be drained retroperitoneally without opening either the pleural or peritoneal cavity The technic of this operation has been described in detail in previous publications[1] [12]. Briefly it consists of resection of the twelfth rib on the right side, following which a transverse incision is made through the soft parts at the level of the spinous process of the first lumbar vertebra. A transverse incision made at this level will prevent injury to the pleura. The incision is carried down to the renal fascia, which is continuous above with the peritoneum. The renal fascia is followed upward to the peritoneum, which is then readily separated from the under surface of the diaphragm. A needle is inserted into the subhepatic region, in order to determine whether an associated subhepatic abscess exists. If no pus is obtained, the separation of the peritoneum from the undersurface of the diaphragm is continued until the abscess on the upper surface of the liver is encountered. By means of the finger the abscess cavity is opened and drained without passing through uninvolved peritoneum or pleura. In this manner an abscess located in the right posterior superior space and also one in the right inferior space may be drained at the same time. In those cases in which an infection of the pleural cavity is suspected a needle may be introduced into the costophrenic angle before opening the subphrenic abscess, in order to determine whether suppuration has occurred in the pleural cavity or not. If such has occurred it is possible to drain the empyema through the same incision and at the original operation by incising the attachment of the diaphragm and the pleura in the costophrenic angle. Cases of empyema unassociated with subphrenic abscess have been treated in this way with very good results by Nather and the author[21]. Two large fenestrated rubber tubes are introduced into the subphrenic abscesses and allowed to protrude through the wound. In cases in which empyema coexists similar tubes are introduced into the pleural cavity in a like manner.

Subphrenic abscesses which point anteriorly, and which may either arise from the "right anterior superior" space or the "left anterior inferior" space, may be drained through the anterior abdominal wall in the manner described by Clairmont[22]. An incision made along the costal margin is carried down to the peritoneum. The peritoneum is then carefully separated from the anterior abdominal wall and the under surface of the diaphragm until the inflammatory mass is encountered. The abscess is opened bluntly. In this way again the abscess may be drained without contaminating an uninvolved peritoneal or pleural cavity. Fortunately in most of the subphrenic infections pointing anteriorly adhesions are already present between the abscess cavity and the parietal peritoneum, a circumstance which facilitates drainage.

INTRA-HEPATIC ABSCESSES.

In considering suppurations within the liver it is necessary to differentiate between the pyogenic and the amebic abscess because of the difference in indications for treatment. Amebic abscess is, fortunately, seen less frequently than previously, due to the efficient anti-amebic treatment, and the treatment of this type of abscess should in reality consist only of prophylaxis. Once a destruction of liver substance has occurred, some type of drainage is indicated. There has been, and still is, considerable controversy about the type of drainage that should be employed in cases of amebic liver abscess. The results obtained by repeated aspiration in the hands of such men as Sir Leonard Rogers[23], Thurston[24], and Manson-Bahr[25] have been, however, much better than any of

those which have been obtained by open drainage. Rogers[23] found in 75 out of 80 cases (86%) of liver abscess at the Calcutta Hospital that the pus obtained from the liver abscess was sterile when the abscess was first opened. On the other hand, cultures made of the pus from the cavity a few days after it had been opened invariably showed secondary infection. He collected 2,661 cases of amebic liver abscess which were treated by the open method, of which number 1,511 died, a mortality of 56.8 per cent. 111 cases were treated by aspiration, together with an amebicide, of which number 16 died, a mortality of 14.4 per cent. The striking change for the worse following open drainage of an amebic abscess in a patient who was not extremely toxic before, is sufficient to make any surgeon hesitate to attempt open drainage of a sterile amebic abscess.

According to Ludlow[26], an amebic, liver abscess is located in the right lobe only, in 93 per cent, in the left lobe only, in 2 per cent, and in both lobes in 5 per cent of the cases. This author recommends aspirating the abscess over the point of greatest tenderness and swelling, which is often the 9th interspace in the anterior axillary line. As soon as pus has been encountered with the aspirating needle, the needle is replaced by a trocar, through which the contents of the abscess may be aspirated. In cases in which the contents cannot be aspirated because of their consistency Ludlow advises the introduction of 20 to 30 c.c. of Dakin's solution. The contents of the abscess are completely evacuated at the time of the aspiration. Subsequent aspirations are performed whenever necessary. Anti-amebic treatment is continued throughout this time.

According to Pannett[27] open operation is indicated: (1), when the abscess is located in the left lobe; (2) when the abscess bulges anteriorly, because of the usual absence of adhesions which will permit a spilling of the contents of the abscess into peritoneal cavity; (3), when aspiration has failed; and (4), when, after aspiration, pyogenic organisms are found in the pus. An additional indication for open drainage may be the localization of the abscess in the dome of the liver, an abscess which cannot be aspirated easily. In such an instance a transpleural approach is justifiable because of the sterility of the contents of the abscess. Rogers[23] found that of his series of cases 40.7 per cent were deeply seated high beneath the ribs. In performing either a transperitoneal or transpleural operation it is essential that contamination of the serious cavity be avoided, even though in the majority of instances the abscess contents are sterile. In cases in which open drainage of an amebic abscess has been performed strict asepsis during dressing of the wound is not only essential but imperative, in order to prevent secondary contamination of the wound with pyogenic organisms. In Rogers' cases in which an open drainage had been performed secondary infection invariably ensued in spite of rigid precautions to prevent such infection.

The treatment of amebiasis is extremely important and essential.

Pyogenic liver abscesses: Aside from amebiasis, the most frequent cause of liver abscess is appendicitis. Liver abscesses, either multiple or single, occur in from .5 per cent to .7 per cent of all cases of acute appendicitis and in about 5 per cent of all fatal cases of acute appendicitis. Infection usually gains entrance to the liver from the appendix or other abdominal viscera by way of the portal vein. Multiple liver abscesses, as a result of portal infection originating in a sigmoidal diverticulitis, have been reported by Whyte[28], Foggie[29], Kramer and Robinson[30]. Unfortunately, following a portal thrombophlebitis numerous radicles of the portal vein within the liver become involved, producing multiple liver abscesses. Occasionally there occur isolated abscesses which may be drained surgically. Brugemann[31], in 1917, found 35 solitary liver abscesses following appendicitis, in

addition to one of his own which he reported. Recently similar instances have been reported by George[32] and Barnes and Pearson[33]. The majority of such abscesses occur in the right lobe. The cause of a solitary liver abscess, following an intra abdominal suppuration and subsequent involvement of the portal vein, is unknown. Quénu and Mathieu[34] believe that an aseptic embolus may reach the liver and produce a focus of necrosis, which may become infected in the course of a bacteremia.

The treatment of solitary liver abscesses is operative incision and drainage. Here, too, as in extra-hepatic abscesses, it is imperative to avoid contaminating uninvolved serous cavities in order to prevent infection of these spaces. A liver abscess, according to its location, can either be drained retroperitoneally, according to the technic described above for subphrenic abscess, or anteriorily. In the latter instance very frequently no adhesions are present between the liver and the anterior parietal peritoneum. In such an instance one may resort to either one or two methods of treatment. At the primary operation the abdominal cavity is carefully packed away from the drainage site, following which the abscess is opened and a tube introduced. Preferably, however, the wound should be packed down to the liver in the region of the abscess for a period of 24 to 48 hours. In this way adhesions will be formed which will wall off the sinus from the general peritoneal cavity. Not infrequently in superficially located abscesses rupture will occur spontaneously following such packing. If, however, this does not occur within 48 hours, the abscess should be opened. The post operative treatment of the pyogenic subphrenic abscess, whether located intra- or extra-hepatically, consists of careful observation of the patient. Dressings should be changed at least once daily and often several times a day. After the discharge has abated one of the fenestrated tubes is removed. Before removal of the second tube it should be clamped for a period of 24 to 48 hours, in order to determine whether symptoms recur or not. As emphasized above, meticulous care must be exercised in the dressing of those cases of amebic liver abscess in which open drainage has been done.

SUMMARY

(1) Subphrenic infections are more difficult to diagnose and treat than other localized intra-abdominal infections.

(2) Subphrenic extra-hepatic infections occur not infrequently during the course of an intra-peritoneal infection. The majority of these cases do not progress to suppuration but subside spontaneously.

(3) Subphrenic extra-hepatic abscess is frequently diagnosed and treated late. The mortality in the past has been high.

(4) Surgical drainage without contaminating an uninvolved peritoneal or pleural cavity will lower the mortality in subphrenic abscess. The mortality in the author's series is 6 per cent.

(5) Amebic liver abscesses are usually sterile. Because of the danger of secondary infection, with its accompanying toxemia, which invariably ensues open drainage of these abscesses, they are best treated by repeated aspirations and the administration of amebicides.

(6) Pyogenic hepatic abscesses usually are multiple and follow a thrombophlebitis of the portal vein. Occasionally solitary hepatic abscesses occur. These, when surgically drained without infecting an uninvolved serous cavity, present a good prognosis.

BIBLIOGRAPHY.

(1) Nather, K. and Ochsner, Alton: Retroperitoneal operation for subphrenic abscess. Surg. Gynec. and Obst., 37:665-673, 1923.

(2) Fifield, L. R. and Love, R. J. McN.: Subphrenic abscess. Brit. Jr. Surg., 13:683-695, 1926.

(3) Bancroft, F. W.: Acute appendicitis. A review of 584 conservative cases. J. A. M. A., 75:1635, 1920.

(4) Stillman, Alfred: Postoperative sequelae in 1748 cases of appendicitis. Med. and Surg. Rep. Roosevelt Hosp., New York, 79, 1915.

(5) Beekman, Fenwick: Acute appendicitis in childhood. A study of 145 cases observed at Bellevue Hospital of New York City. Ann. Surg., 79:538, 1924.

(6) Clairmont, P. and Meyer, M.: Erfahrungen ueber die Behandlung der Appendicitis. Acta. Chir. Scand. 60: 55, 1926.

(7) Cutler, C. W.: Postoperative complications of suppurative appendicitis. Am. Jr. Surg. N. S. 3:603, 1927.

(8) Suermondt, W. T.: Resultate der chirurgeschen Behandlung der Appendicitis beurteilt nach den letzen 1000 Faellen der chir. Klinik zu Leiden. Deut. zeit. f. Chir., 170:289; 1922.

(9) Neuhof, Harold: Experimental ligation of the portal vein; its application to the treatment of suppurative pylephlebitis. Surg. Gynec. and Obst., 16:481, 1913.

Idem: Non-suppurative subphrenic peritonitis complicating appendicitis. Surg. Gynec. and Obst., 14:231, 1912.

(10) Lee, Roger I.: Subdiaphragmatic inflammation. J. A. M. A., 64:1307-1310, 1915.

(11) Clendening, L.: Subphrenic infection. Med. Clin. N. A., 7:1147-1167, 1924.

(12) Ochsner, Alton: Subphrenic abscess. New Orleans M. and S. J., 81:102-111, 1928.

(13) Douglas, J.: Subdiaphragmatic abscess and accumulations of fluid. Am. Surg., 79:845-853, 1924.

(14) Hodges, F. M.: Subdiaphragmatic abscess. J. A. M. A., 80:1055-1058, 1923.

(15) Eicher, C. G. and Kibzey, A. T.: Subdiaphragmatic abscess. Atlantic M. J., 28 30-32, 1924.

(16) Bauman, M.: Subphrenic abscess. Beitr, z. klin. Chir. 128:477-480, 1923.

(17) McEachern, J. D.: Closed drainage in subphrenic abscess. Surg. Gynec. and Obst., 43:215-219, 1926.

(18) Tuft, L.: Subphrenic abscess, clinical study. Am. J. M. Sc., 170:431-441, 1925.

(19) Lotsche, F.: Subphrenic abscess. Klin. Wchnschr. 3:2013-2014, 1924.

(20) Lockwood, A. L.: Subdiaphragmatic abscess. Surg. Gynec. and Obst., 33:502, 1921.

(21) Nather, Karl and Ochsner, Alton: The resection of the twelfth rib as an operation for total empyema. Surg. Gynec. and Obst., 39:94-99, 1924.

(22) Clairmant, P.: (Quoted by Ochsner [12]).

(23) Rogers, Sir Leonard: Amoebic liver abscess, its pathology, prevention, and cure. Lancet, 1:463-569, 1922.

(24) Thurston: A series of 101 cases of abscess of the liver. Indian Med. Gaz., 49:88, 1914.

(25) Manson-Bahr: Treatment of liver abscess by aspiration. Sancet, 1:941, 1923.

(26) Ludlow, A. D.: Liver abscess. Report of 100 operations. Surg. Gynec. and Obst., 36:336, 1923.

(27) Pannett, C. A.: Solitary abscess of the liver. Clin. Jour., 49:261, 1920.

(28) Whyte, J. M.: A case of suppurative hepatitis following inflammation in an acquired diverticulum of the sigmoid flexure. Scottish M. and S. Jr., 18:120-123, 1906. (Quoted by Foggie).

(29) Foggie, W. E.: A case of diverticulitis with metastatic suppuration. Lanet, 1:1357, 1921.

(30) Kramer, S. E. and Robinson, W.: Acquired suppurative diverticulitis with pylephlebitis and metastatic suppuration in liver. Surg. Gynec. and Obst., 42; 540, 1926.

(31) Brugemann, H. O.: Isolated abscess of the liver complicating appendicitis. Ann. Surg., 65:462, 1917.

(32) George, Wm.: Appendicitis and hepatic abscess. Brit. Med. Jr., 1:633, 1928.

(33) Barnes, W. A. and Pearson, L. V.: Suppurative pylephlebitis and hepatic abscess complicating appendicitis. Brit. Med. Jr., 1:390, 1928.

(34) Quenu, E. and Mathieu, P.: Rev. de Chir. 44: 519, 1911.

DISCUSSION OF THE PAPERS OF DRS. MUSSER, GRANGER AND OCHSNER.

Dr. J. G. Snelling (New Orleans): In this series of cases that has just been reported by Doctors Granger, Musser and Ochsner, I happen to have had two under my direct supervision, and I just want to mention a few of the facts to lend my say to the method that has been discussed and to some of the points that have been brought out.

This last case Dr. Granger showed us, a little boy who is now in Ward 158, was seen in consultation in a pediatric service. However, after roentgen-ray findings by Dr. Granger, and together with the laboratory and clinical findings, a rather positive diagnosis of liver abscess was made. The boy was very septic, ran a temperature of 103° to 104° daily and was quite run down, as he would be after a week or so of this condition. The previous history in this case was not as good as we have sometimes gotten. However, I gleaned from the mother that the child had had, about seventeen days previously, an attack of cramps in the abdomen and some little diarrhea, which lasted for two or three days and then he was taken with a chill and higher fever and pain in the upper right part of abdomen. He was brought to the hospital with the tentative diagnosis of possible empyema, and later on a possible liver involvement was considered. Subsequently, the diagnosis was made as I have stated.

This child I had the opportunity of operating on, and used the method just described by Dr. Ochsner. I have had occasion to use this procedure on about six cases in the last two years, four of which were pyogenic and two of the solitary type. One of the pyogenic cases died, but autopsy in this case showed multiple liver abscesses, one of the largest of which we had been successful in draining. I am counting the case under discussion as one of recovery, because at the present time he is doing so nicely, having been temperature free for several days now.

It illustrates very nicely the point brought out by Dr. Granger in showing the obliteration of the costophrenic angle, and with diminution but not obliteration of the cardio-hepatic angle in the liver abscess. This child I operated on for liver abscess and approached through the bed of the twelfth rib. I was able to aspirate a small amount of pus, just enough to get into the aspirating needle, about two inches within liver substance. I could feel the needle plunge into the abscess cavity by the difference in density. Going on this finding then I entered the liver with blunt dissection and evacuated about six ounces of thick greenish yellow pus, proving to be a liver abscess. The little chap was sent to the ward and seemed to improve; however, his temperature did not drop to normal. I was then confronted with the possibility of why he did not improve more rapidly. I considered the possibility of multiple abscess of the liver and the possibility of septicemia. We had blood cultures made which were negative and strengthened the child with treatment—transfusion among other things. After procrastinating for eight or ten days, I decided to call the Roentgen-ray Department in for further consultation and we got the two pictures which Dr. Granger showed. I was then able to go back through the same incision, there being only granulation tissue present, and by blunt dissection I was able to break into a large sub-diaphragmatic abscess in the posterior space. That fits in with the occurrence in the posterior space in cases following disturbances in, or some infection of the gastro-intestinal tract. One other point which I think is illustrated is the fact that here was a child who was extremely septic and any any other involvement would have been enough to push him over the brink. Having this tract there to go through, and being able to avoid both pleural and peritoneal cavities, was decidedly in his favor. Immediately after evacuation of the subdiaphragmatic he picked up and looks as though he will be well in quite a short while.

Dr. Urban Maes (New Orleans): I am called upon to do rather an impossible thing, namely, to contribute something to a subject about which the essayists, it seems to me, have already said all that there is to say.

Most experienced surgeons have no difficulty in making a diagnosis of suppurative conditions in and about the liver, because, as a rule, they are called to see the patients when the pathology is definitely established. We would be called earlier, I think, if the aphorisms were remembered more frequently that in continued fever of undetermined origin, it is a wise plan to suspect the liver. I can testify that I owe several successful early diagnoses of my own to the recollection of this fact.

Authors differ surprising in the percentages they assign to the various causes for subphrenic abscess or suppuration about the liver. One of the older writers, I remember, considers 80 per cent of them due to gastric or duodenal ulcer. Others are inclined to consider septic processes in the abdomen as a more frequent cause. In the opinion of others age has something to do with the incidence, and it is Aschoff, I think, who states that because of the active lymphatic system in young subjects they most frequently exhibit a diffuse purulent peritonitis, while in older people the condition, like appendicitis, is of vascular origin. Indeed, in old people appendicitis is a frequent precursor of liver abscess, giving us the typical appendicular liver described by DeBovis and others of the French school.

In previous years, because I knew of no better way, it was my custom to use the trans-pleural approach, with a special technic that prevented the spill of pus into the peritoneal cavity. This method was sufficiently satisfactory for single abscesses but was not always safe for multiple ones, and I have gladly substituted for it the approach described by Dr. Ochsner, who, with his cutomary modesty, has omitted to say that he devised the technic himself.

A great proportion of the amebic abscesses of the liver are in the upper right lobe, and the reason for this has been clearly shown by Counseller and McIndoe of the Mayo Clinic: the right and left lobes of the liver are separate and distinct, their blood vessels do not communicate, and the larger portion of the blood supply is carried by the portal system directly to the right lobe. Hence the high incidence of infections in it, and the comparative immunity of the left lobe.

Amebic abscesses of the liver frequently appear without the classic preliminary symptoms of dysentery, diarrhea or bowel disturbance, and these cases, even when seen late, may be difficult to recognize, unless, as I have already pointed out, it is remembered that it is well to suspect the liver in all instances of continued fever of undetermined origin. Previous disturbances of the gastro-intestinal tract due to amebiasis are not always exhibited.

In these cases it is frequently necessary to get rid of a large amount of débris, so that the aspirating needle is entirely unsatisfactory and surgical management is necessary. It seems rather futile to take issue with such statistics as those of Rogers in regard to emptying the abscess with the syringe or the trocar.

These patients should not be dressed very frequently. It is my custom to fill the abscess cavity with some sterile material, preferably

balsam of Peru, with a sterile packing, and not to disturb the wound for at least a week. Every time it is disturbed, there is the risk of introducing fresh infection, and I have had no occasion, in the cases I have handled in this way, to regret my inactivity. Daily dressings are definitely contra-indicated.

Dr. George Herrmann (New Orleans): My experience with subdiaphragmatic abscess has been considerably limited. Within the last two months, however, I have had occasion to observe two cases in which the diagnosis was considered, but in which the proof was much more difficult to obtain than has been indicated this evening. The question always arises as to whether the process is above the diaphragm or below the diaphragm when both chest and abdominal symptoms present. The tendency is always to adhere to the clinical rule of considering one process responsible for the symptoms in both regions. It must not be forgotten that we may have totally unrelated pathological processes above and below the diaphragm on the same side. This was forcefully brought home to me this morning. One of our patients had a very definite pleurisy with a friction rub in the left axilla. He also had a palpable mass in the left upper quadrant, slight rigidity, tenderness, and fever of more or less septic type. Salicylates had been given for his chest condition and the temperature curve was modified. It was only after repeated questioning that he gave a definite history of regularly occurring chills and fever some weeks previously. Examination of the blood showed estivo-autumnal malarial parasites. These, of course, accounted for the mass enlarged spleen in the left upper quadrant.

Another patient, who is also in our ward at present, came in because of an attack of acute pain in the left upper quadrant, chills, and fever. He gave a very definite history of exposure to rain and cold, following which his pain in the upper left chest was noted. He also presented rigidity and tenderness of the left upper quadrant, considerable distention, and tenderness around the costoveterbral angle on the left.

The roentgenologist was non-committal and made a diagnosis of diaphragmatic pleurisy in this case. There was very definite evidence of compression at the left base, dullness and distant breath sounds and rales. Here, again, we were faced with the question of whether we were dealing with one process or two.

Urinalysis showed pus in the urine. On the basis of this we considered the presence of an infectious or surgical kidney with a paranephric abscess that had become subdiaphragmatic. The chest signs persisted as has also the fever. Exploratory puncture had been advised, but we hesitated because of the danger of undertaking such procedure.

These two cases seem to me to emphasize the fact that the diagnosis is by no means as simple as one might be led to believe. It is true that in the classical case, the symptomatology and clinical findings are clearly diagnostic as they have been presented, but it is rare, in my experience, at least, to find these processes of the classical type. When there is a question of subdiaphragmatic abscess, although an internist, I prefer to have a surgeon make an open exploratory incision rather than to blindly stick a needle into a body cavity where there is a possible pus cavity that is well walled off.

I would conclude by reiterating that it is quite possible, I believe, to have two distinct pathological processes—one above and one below the diaphragm on the same side; and the rules of always considering one diagnosis and one condition as accounting for both processes should not be iron bound.

Dr. C. W. Duval (New Orleans): I do not know that there is anything I can add, so thoroughly has the suject been covered by the essayists; however, it may be of interest to say a few words concerning the nature and route of infection in hepatic and perihepatic abscesses. Amebiasis and the pyogenic infections of the liver and the perihepatic space are secondary to a primary infection of the intestinal tract. Never in the amebic and rarely in the pyogenic are the liver lesions secondary to a pre-existing bacterimia.

The route by which the infectious agent is conveyed to the liver and to the region about the liver, is the portal vein or the lymphatics. Dr. Maes, quoting Aschoff, states that in the very young the infection travels to the liver by the way of the lymphatics, while with older subjects it goes by the veins. I doubt that age is a factor in determining the route of infection. Of course, it is quite evident that in all cases the metastasis either is through the lymphatics or veins because otherwise we would not get colonization of the infectious agent confined to the liver.

The kind of pathogenic agent is a factor of considerable importance from the standpoint of treatment and prognosis. More commonly subdiaphragmatic abscess is due to the colon bacillus and only rarely caused by the staphylo-streptococcal groups. When the infection is due to the latter it seldom remains localized but becomes a general infection which is not amenable to surgical intervention.

If ameba is the infecting agent it is well to bear in mind that the lesion produced in the liver is commonly solitary. Small multiple abscesses of the liver are almost always pyogenic. Though

both infections are characterized by what is termed an abscess, the amebic lesion is wrongly called because it is necrosis and not an acute inflammatory condition. In liver amebiasis it is only an abscess when there is a superimposed secondary pyogenic invasion of the amebic lesion. It is generally thought that the hepatic lesions in amebiasis is sterile in so far as pyogenic bacteria are concerned, and only after the so-called abscess is opened and drained does it become infected. While it is true that drainage permits of extraneous germs entering, we must not lose sight of the fact that the liver lesion is from the beginning a mixed infection. This is explained on the ground that the liver normally contains bacteria that are constantly passing through from the intestinal tract by way of the portal circulation. Such bacteria also account for the occasional purulent character of the necrosis in hepatic amebiasis.

Dr. J. Birney Guthrie (New Orleans): I feel that the opinion of everyone here tonight is that we have all been amply repaid for coming in listening to this symposium. I was a little disappointed to hear of the change in title of Dr. Musser's paper. "The Thoracic Physical Signs of Liver Abscess" was a title that appealed strongly to me.

As the diaphragm is pushed up, contrary to what we would expect, any change in density of the lungs is not manifest at the base, but at the apex of the lung. In this case we see by the radiograph that the right lung has been moved forward and upward by the underlying liver enlargement. Base of lung is pushed forward. We also see a change in the position of the heart which is pushed to the left also by the upward encroachment of the liver.

The alteration of the position of the heart's apex excepting as indicates often a change in the neighboring organs rather than a change in the heart itself.

In throacic diagnosis, the determining of the position of the cardial apex may lead us to the finding of some cause as in this case relatively remote from the heart.

Dr. Allen Eustis (New Orleans): Dr. Ochsner very correctly stated that the vast majority of cases of abscess of the liver which come to a surgeon are of long standing, and have apparently been neglected. This, however, should not be taken as a criticism of the medical man, as the diagnosis is often fraught with great difficulty.

If the abscess is confined to the liver only, the individual is often treated for typhoid fever; on the other hand, if the abscess perforates the diaphragm, pulmonary symptoms predominate, and even after rupture into the bronchus, this type is frequently treated as tuberculosis. During my experience in rural practice I saw a great many cases of amebic abscesses of the liver, and it was often necessary to make the diagnosis without the aid of the roentgen-ray, which of course, at the present day simplifies the diagnosis very considerably.

In 1919 I reported the physical signs in this type of case, which I consider is pathognomonic of the condition, namely: bronchophony and pectoriloquy over the liver.

Dr. D. L. Watson (New Orleans): Will you excuse me, a family physician, attempting to offer any suggestion to the splendid discussions we have had here this evening? But abscesses of the liver due to amebae must be diagnosed from abscesses due to other causes.

The discussion has shown that about 80 per cent of all abscesses of the liver are either sterile or amebic. As a rule a sterile abscess is amebic whether the aspirated fluid shows it or not. Surgical procedure in amebic abscess is very dangerous and should be avoided if possible. The differential diagnosis is made by giving one grain emetine hydrochloride intravenously daily for about ten days and less often thereafter. The gradual improvement of the patient will eliminate other causes for the abscess. Emetine thus given will prove a specific for the abscess.

Emetine hydrochloride intravenously is a specific for amebiasis in any form.

Dr. A. L. Levin (New Orleans): It might be of interest to the members present here tonight to mention a point that was not discussed in connection with liver abscess; that sometimes presence of pus in the liver will give rise to a positive Wassermann reaction as a result of the lipoid bodies present in the blood stream. A mistaken diagnosis of syphilis will be the result.

I reported a similar case about 15 years ago. A middle aged man developed upper abdominal pain and temperature which persisted for months. He was treated for paratyphoid. On my first examination, I noticed a bulging in the upper right abdomen, and on palpation there was a mass of doughy consistency. I advised him to see me the following morning for a gastric analysis. During the night, he applied hot poultices to the painful area. On the second examination, the mass had disappeared. The stomach tube revealed a quantity of pus and blood in the stomach. A roentgen-ray of the G. I. tract revealed definitely a double track leading into the stomach from the liver area. Every laboratory in the city gave a 4 plus positive Wasserman on the patient, for many months. Heavy doses of mixed treatment did not influence the positive Wassermann

reaction. A liver abscess was finally discovered in the left lobe of amebic origin. Abscess was drained surgically and patient got perfectly well. The blood Wassermann became negative.

Dr. John Musser (closing): Gentlemen, the hour is late. I shall say only a few words.

I am grateful to Dr. Eustis and Dr. Jamison for giving the diagnostic suggestions they have. I wish to say I appreciate the courtesy of those who so kindly entered in the discussion of this symposium.

Dr. Amedee Granger (closing): I would just like to emphasize the importance of the lateral view. I feel quite sure that there are some cases such as those Dr. Jamison spoke of when it might be quite difficult to decide whether the pathology is below or above the diaphragm. Two plates should always be made because the lateral view is extremely important. I can recall numerous instances when with a P. A. view alone the diagnosis was quite impossible and a lateral view cleared it up; and although I admit the possibility of failure, I do believe failure will be reduced to a minimum if lateral views are made. These should be made at a target-plate distance of six feet or more, with the patient sitting or standing. When the patient is lying down any fluid content flows over the entire pleural space and it can no longer be diagnosed positively.

Dr. Alton Ochsner (closing): In regard to Dr. Maes' remarks and his discussion of the technic which he has used, I believe that if it is possible to prevent secondary infection in this way that open drainage of amebic abscess is the method of choice, especially in those cases in which the abscess is located high in the lover.

In regard to Dr. Eustis' statement, I meant to imply that the pyogenic infections and not the amebic abscess were often diagnosed late by the surgeon. The criticism in this instance is due the surgeon himself as usually the patient is in the surgical service at the time this complication develops.

I grant that the type of case which Dr. Jamison described presents a real difficulty. The dianosis in this instance is much more difficult than in the case in which the subphrenic infection develops in the hospital, the course of which can be followed.

I was pleased that both Drs. Musser and Granger failed to mention the presence of a gas bubble immediately below the diaphragm as a diagnostic point in subphrenic infections. In practically all textbooks this finding is mentioned and considerable stress is placed upon it. In reality it is seldom found and when found, it is almost invariably a late finding.

Concerning Dr. Watson's experience with emetine in the treatment of amebic abscess, I wonder if in some of his cases the patient had an amebic hepatitis and not a true abscess. From physical findings it is often difficult to distinguish between hepatitis and abscess formation. The former, however, responds well to the use of amebacides, whereas the latter does not. For this reason all cases suspected of being amebic abscess should be treated with amebacides before any surgical intervention is attempted.

SOME DIAGNOSTIC AND PROGNOSTIC BLOOD INDICATIONS.*

LEON S. LIPPINCOTT, M. D.,

VICKSBURG, MISS.

The blood, briefly, is made up of red and white corpuscles and platelets floating in a clear, straw-colored fluid, the plasma. The white corpuscles or leukocytes are cells derived from parent cells situated in certain formative organs of different types. The leukocytes, as found in the circulating blood, are of two general types—those containing granules in their cytoplasm and those without true granules.

In the normal adult, granular leukocytes are formed in the bone marrow, and according to the staining reaction of their granules by Wright's Stain, whether purple, red, or dark blue, are divided into neutrophils, eosinophils, and basophils. All when mature have rather complex lobulated nuclei and in normal blood most granular leukocytes are mature.

In normal blood, there are a few neutrophilic cells (about four per cent) without lobulation of the nuclei, although the nuclei are bent and indented. These are metamyelocytes and represent a stage in the normal development of the more complex polymorphonuclear cells. In other words, they are immature neutrophils.

In some pathological conditions, there appears in the blood a type of neutrophilic cell found normally only in the bone mar-

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row. This is a mononuclear rather than a polymorphonuclear cell, the myelocyte, or fully granular precursor of the metamyelocyte and polymorphonuclear leukocyte.

In the bone marrow in health are some cells with simple round nuclei and with few granules in their cytoplasm. These are the premyelocytes, and are the precursors of the myelocytes. The premyelocytes are derived from non-granular ancestors, the myeloblasts.

In brief, the life history of a neutrophilic polymorphonuclear leukocyte begins in the bone marrow in a simple cell with round nucleus and no granules. This cell then gradually develops granules in its cytoplasm and the nucleus becomes lobulated and more and more complex until maturity in the blood stream.

The eosinophilic polymorphonuclear leukocyte and the basophilic polymorphonuclear leukocyte develop in a similar way with life histories identical with those of the neutrophil.

In normal blood the leukocytes without true granules are of two kinds, the monocytes and the lymphocytes. The monocytes or endothelial leukocytes or large mononuclear leukocytes, as they are sometimes called, have an uncertain origin about which there has been much discussion. These cells are larger than the polymorphonuclear leukocytes and the nuclei are round or indented. The nuclei are basophilic, the cytoplasm faintly basophilic. The lymphocytes originate in the lymphatic tissues scattered throughout the body. Most of them are small and show only a slight rim of protoplasm about round or slightly indented nuclei, which are usually eccentric. Larger forms occur, most frequently in children, and are now generally considered as young forms of the small lymphocytes. The lymphocytes are basophilic, the nuclei staining much more intensely than the cytoplasm.

LEUKOCYTOSIS.

An increase in leukocytes, or leukocytosis, occurs in most infections at some stage. Leukocytosis in acute cases depends almost entirely upon an increase of the neutrophilic granular cells. At the same time, there is generally a reduction in the eosinophilic cells. There are important changes also in the qualitative blood picture.

These changes are principally in the degree of maturity of the cells. The appearance of young, immature neutrophilic cells in the circulating blood—an indication of an emigration of young, immature neutrophilic cells from the marrow, is believed to indicate active infection, and the younger and the more immature the cells are, the more active the infection. This is explained by the fact that the neutrophilic cells form the main line of resistance to most infections. When an infection occurs, the marrow puts into the circulation more neutrophilic cells to combat it. There is a moderate store of mature cells at first to account for the increase found. If the infection is not at once overcome, however, the call on the marrow is greater than can be supplied with mature cells, and the marrow then puts into the blood stream cells that have not had time to reach maturity. The greater the call, the younger the cells that must be sent out to meet it.

Numerous "indices" have been proposed to bring out and apply these qualitative changes in the blood to the conditions underlying. The simplest and most practical index is that of Schilling, and from it much valuable information may be derived. Schilling divides the neutrophilic cells into groups as follows: Myelocytes; young metamyelocytes (with only the slightest indentation of the nuclei); older metamyelocytes (with deep indentation, but no true lobulation of the nuclei); and polymorphonuclears. In making the ordinary differential count, each of these types is counted separately, so that each

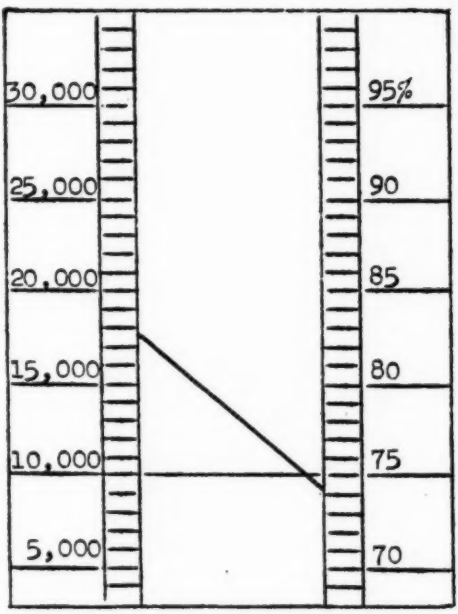
variety is expressed as a percentage of the total number of leukocytes. The neutrophilic cells are thus arranged in order of maturity. In our present work, we have grouped the young metamyelocytes and the older metamyelocytes together under the term, "immature forms."

Schilling, according to Piney[1], gives the following as normal:

Total leukocytes 6,000 to 8,000 per cu. mm.	
Lymphocytes	23 per cent
Monocytes	6 " "
Neutrophils:	
Young metamyelocytes	0 " "
Older metamyelocytes	4 " "
Polymorphonuclears	63 " "
Eosinophils	3 " "
Basophils	1 " "

In our experience in this locality 23 per cent is slightly low for the lymphocytes and 3 per cent is rather high for the eosinophils in normal persons. Otherwise, what we consider average normal counts are very similar to the normal of Schilling.

Todd and Sanford[2] call attention to the fact that in infectious and inflammatory conditions, a comparison of the percentage of neutrophilic cells with the total leukocyte count yields more information than either alone. They quote Sondern that the percentage of neutrophils represents the severity of the infection or the degree of toxic absorption, and the total count indicates the patient's power of resistance. In moderate infections, with good resistance, the leukocyte count and percentage of neutrophils will be increased proportionately. When the neutrophilic percentage is increased to a greater ex-



GIBSON CHART.

White, male, age 14.
Admitted Sept. 18, 1928.

Severe pain in pit of stomach began morning before admission; nausea and vomiting. Pain moved to lower abdomen and appendix region later in day.
Temp. 99° F. Pulse 83.
Abdomen rigid; tender over region of appendix.
Operation Sept. 19, showed free straw-colored fluid in peritoneum; appendix retrocecal; large gangrenous tip; fibrinous exudate.
Microscopic examination showed acute purulent inflammation.

Immature Neutrophils 28%

Temp. 100.6° F. on Sept. 20; normal on and after Sept. 22.

ACUTE APPENDICITIS (Case 1).

tent than is the total number of leukocytes, no matter how low the count, either very poor resistance or very severe infection is indicated.

By means of an arbitrary chart suggested by Gibson and shown in the work of Todd and Sanford, this relationship may be expressed graphically and is an aid in interpreting counts. An ascending line from left to right indicates an unfavorable prognosis in proportion as the line approaches the vertical. All fatal cases show a rising line. A descending or horizontal line suggest a favorable prognosis.

By an application of the Schilling index and the Gibson chart, the surgeon can obtain much valuable information as to indication for operation, the time of operation, and the prognosis in operative cases. The blood findings in some clinical cases encountered during the past year will serve to illustrate their value:

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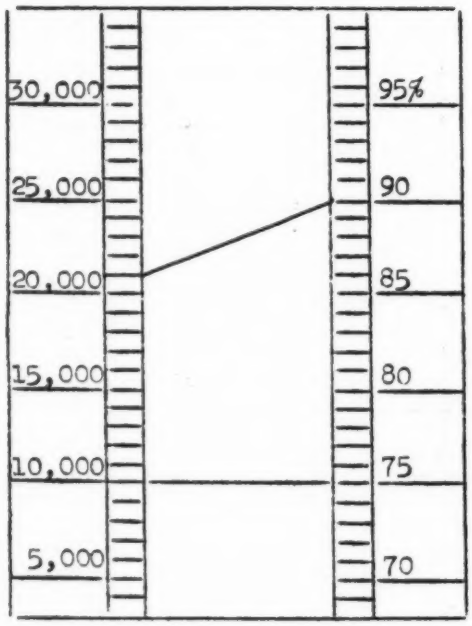
1. Acute appendicitis—Temperature 99°F.; pulse 83, total leukocytes, 17,600; neutrophils, 74 per cent, of which 28 are immature. A definite infection but with a descending line on the Gibson chart, indicating good resistance. Rapid recovery after operation; temperature normal in two days.

2. Acute appendicitis—Temperature 100.4°F.; pulse 75. Total leukocytes, 21,000; neutrophils 90 per cent, of which 64 are immature. More severe infection than in No. 1, and with a rising line on the Gibson chart, indicating poor resistance. Microscopic examination of appendix showed gangrene. Temperature was normal in six days.

3. Acute appendicitis—Temperature 99.8°F.; pulse 96. Total leukocytes, 14,600; neutrophils, 82 per cent, of which 40 are immature. Moder-



GIBSON CHART.

ACUTE APPENDICITIS (Case 2).

Immature Neutrophiles 64%

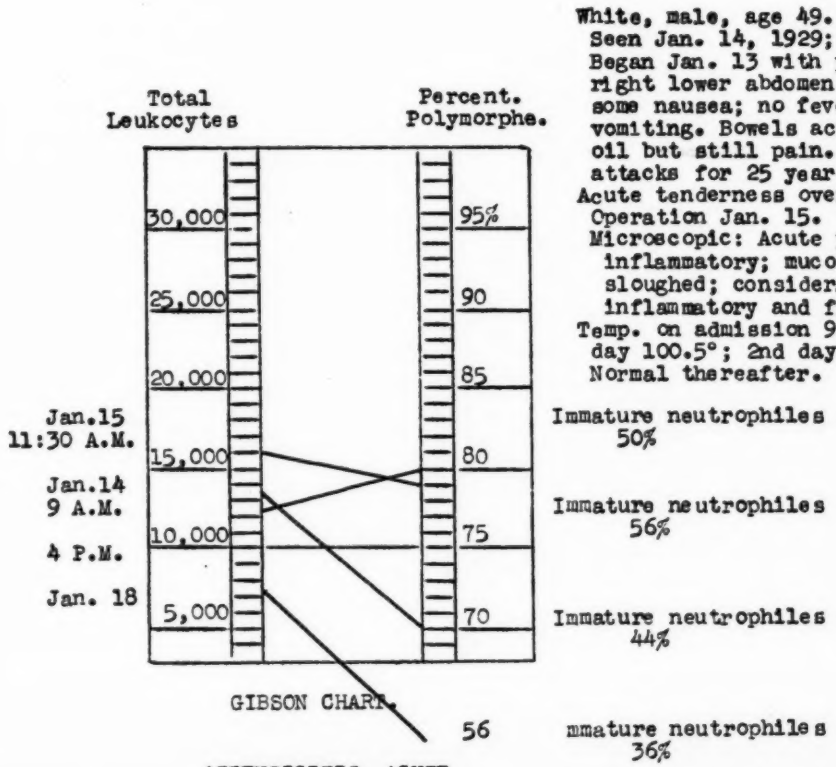
White, female, age 34.
Admitted Nov. 9, 1928.
Pain in right lower quadrant; Temp. 100.4° F. Pulse 75. Abdomen soft; no masses; some tenderness over appendix.
Immediate operation showed cecum bound down and bent on itself so that appendix was fixed to cecum and colon high up under hepatic flexure.
Microscopic examination acute inflammatory; mucosa partially sloughed.
Temp. was 101° F. day following operation; reached normal sixth day postoperative.

ately severe infection; rising line—resistance not good. Appendix gangrenous. Temperature normal in four days after operation.

4. Acute appendicitis occurring in chronic appendicitis of many years duration. Symptoms similar to numerous previous attacks. On January 14 at 9 a.m.—leukocytes, 13,300; neutrophils, 70 per cent, of which 4 were immature, indicating a definite infection with good resistance. At 4 p.m. of the same day, however, the blood picture showed a distinct change for the worse, with leukocytes 12,200 and neutrophils, 80 per cent, with 50 immature forms, and with a rising line on the Gibson chart. The next morning the leukocytes were 16,000 and the neutrophils 79 per cent, showing infection more severe as indicated by rising total count and immature forms 56, but with better resistance than the night before. Operation was performed and temperature became normal three days later. The blood picture at this time was also much approved.

5. Acute appendicitis and acute right salpingitis occurring in a chronic condition. Leukocytes were 10,700; neutrophils 75 per cent. This picture by itself with moderate fever did not give definite information, but when it was considered that 34 of the neutrophils were immature forms, infection was indicated. The next day the temperature was normal, but the leukocytes were 9,600 with 80 per cent neutrophils and 52 immature forms, a rising line on the Gibson chart, showing resistance was being lost. Temperature was normal six days after operation.

6. Chronic appendicitis, with slight acute inflammatory—Leuckoytes 15,300, with 81 per cent neutrophils and 50 immature forms. Definite infection with fairly good resistance. Patient had had several previous similar attacks and recovered without operation. The next day leukocytes were 10,600; neutrophils 73 per cent; immature neutrophils 43; eosinophiles 1 per cent, indicating considerable improvement and condition better. On the day following operation, leukocytes were 12,800 with 72 per cent neutrophils and 44 immature forms, showing good resistance. Temperature was normal five days after operation.



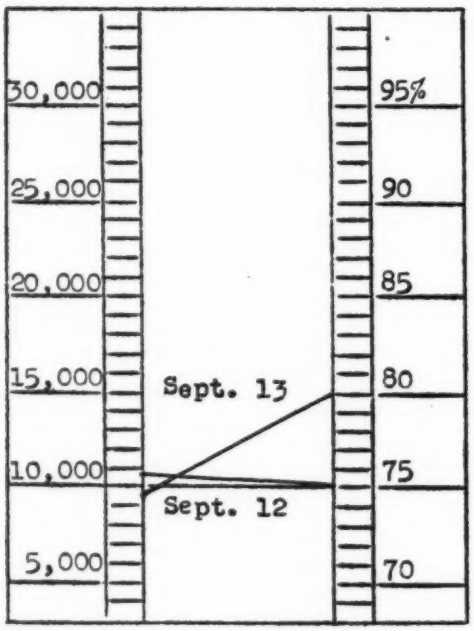
APPENDICITIS, ACUTE
APPENDICITIS, CHRONIC
(Case 4).

7. Patient admitted with symptoms of acute appendicitis; temperature 99.8°F.; pulse 90. Leukocytes were 5,600 with neutrophils 52 per cent and 1 immature form. The next day leukocytes were 6,200; neutrophils, 58 per cent; 3 immature forms; eosinophils, 1 per cent. No evidence of infection and Gibson chart showed a descending line at both examinations. Operation showed cystoma of the right ovary with twisted pedicle; appendix showed slight chronic inflammatory change.

8. Acute choleoystitis—Temperature 88.4°F.; pulse 84. Leukocytes, 21,200; neutrophils 89 per cent, with 48 immature forms. Decided infection and moderately rising line. The next day leukocytes were 29,900; neutrophils still 89 per cent; but with only 8 immature forms. This was a decidedly better picture in spite of the higher total count. Temperature never went over 100° following operation.

9. Acute cholecystitis; cholelithiasis—Temperature 102°F.; pulse 96. Leukocytes, 26,200; neutrophils, 89 per cent; immature forms, 55. Definite, rather severe infection; good resistance. Temperature normal five days after operation.

10. Impacted common duct stone; gall bladder removed 9 years before. Temperature, 100°F.; leukocytes, 12,700; neutrophils, 94 per cent; immature forms 70. Severe infection and poor re-



White, female, age 28.
Admitted Sept. 12, 1928.
Pain in right lower quadrant for one week, requiring morphine for relief.
Temp. 102° F. Right lower abdominal tenderness; tender mass in right side of pelvis.
Operation Sept. 14.
Temp. not over 100.2° F. after operation; normal after Sept. 20.
Microscopic examination showed considerable chronic inflammatory in addition to the acute condition of appendix and tube.

Immature Neutrophiles 52%

Immature Neutrophiles 34%

Temp. was normal on Sept. 13.

GIBSON CHART.

ACUTE APPENDICITIS
CHRONIC APPENDICITIS
SALPINGITIS, CHRONIC AND ACUTE, RIGHT
CYSTOMA OF RIGHT OVARY
(Case 5).

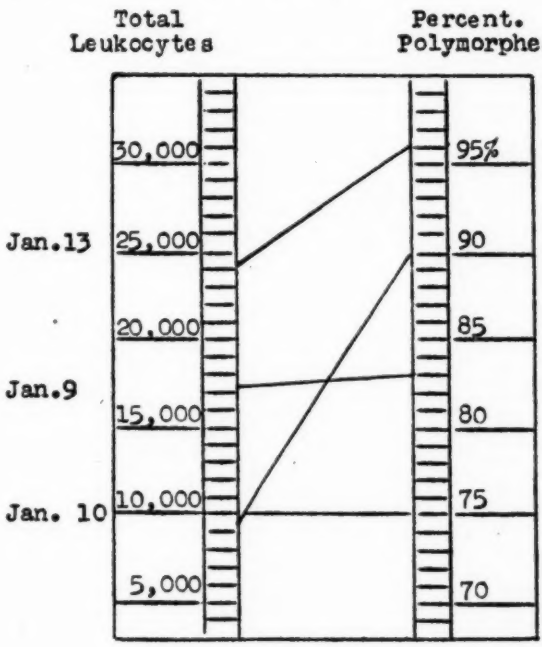
sistance. The next day picture was much improved, with leukocytes 19,900; neutrophils, 80 per cent; immature forms, 56. Temperature was normal two days after operation.

11. Acute bilateral pyelitis in a child. Irregular temperature for a month. On October 23, leukocytes were 18,000; neutrophils, 80 per cent; immature forms, 52. October 25—leukocytes, 24,300; neutrophils 84 per cent; immature forms, 64. October 26—leukocytes, 21,000; neutrophils, 79 per cent; immature forms, 46. November 4—leukocytes, 33,900; neutrophils, 84; immature forms, 50. Blood picture showed definite severe infection throughout but good resistance at all times. This case illustrates the fact that the blood usually shows a more definite reaction in pyelitis in children than in adults. Compare with next case.

12. Acute right pyelitis and cystitis in an adult female. Temperature 98° to 99°F. Leukocytes, 8,600; neutrophils, 75 per cent; immature forms, 32 per cent. The total leukocyte and neutrophil counts alone do not give any definite information in this case except that the Gibson chart shows a slightly rising line. The immature neutrophils together with the finding of urinary infection were of value in establishing the cause of illness and its symptoms.

13. Acute pyelitis and chronic cystitis in an adult male eight weeks after prostatectomy. Illness of five days' duration with temperature of 104° F., which dropped under quinin treatment. Temperature on admission 102.6°F.; pulse 90. Leukocytes were 11,000; neutrophils, 75 per cent; immature forms, 52, indicating moderate infection and good resistance. Malaria had to be considered but the blood picture was not usual for that condition. The urine showed pus and colon bacillus infection.

14. Abscess of the lower jaw following difficult wisdom tooth extraction. Temperature 102.8°F. to 103°F.; pulse 84. August 27—leukocytes, 12,000; neutrophils, 79; immature forms, 33, indicating definite infection but rather poor resistance. August 28—leukocytes, 15,000; neutrophils, 89 per cent; immature forms, 50, a picture indicating condition definitely worse. Under treatment, temperature was normal August 31, and patient discharged.



GIBSON CHART.

BRONCHO-PNEUMONIA

(Case 22).

15. Cellulitis of right arm. Temperature 102° F.; pulse 120. Leukocytes, 19,000; neutrophils, 77 per cent; immature forms, 51. Severe infection with good resistance. Irregular temperature for 10 days.

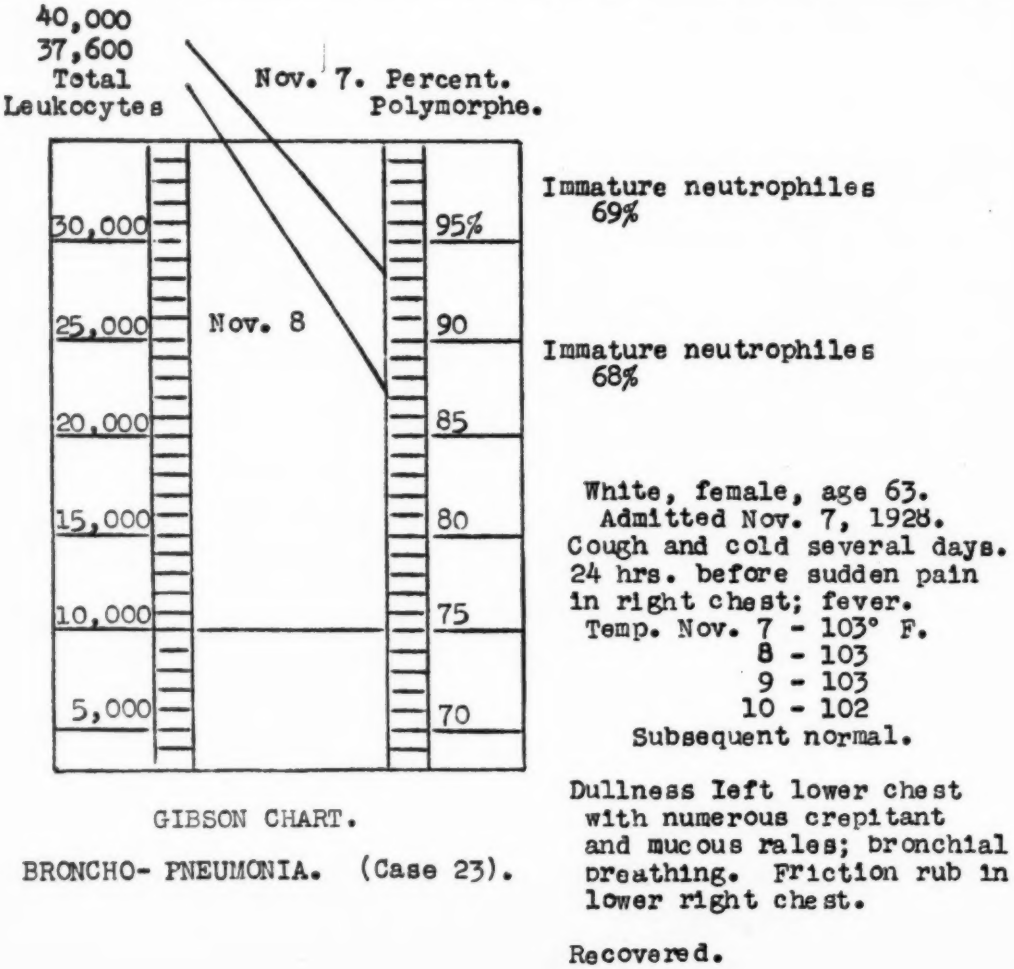
16. Mastoiditis, acute. Temperature 99°F.; pulse 120. Leukocytes 19,800; neutrophils, 81 per cent; immature neutrophils, 59. Definite pus infection; good resistance. Irregular temperature, 99 to 102°F., for 6 days following the operation.

17. Mastoiditis, acute; otitis media, acute and chronic. Temperature 99°F.; pulse, 78. Leukocytes, 13,000; neutrophils, 78 per cent; immature forms, 35. Definite infection; good resistance. Irregular temperature, 97° to 103°F., for 12 days following operation.

18. Multiple sinusitis. Temperature 99°F.; pulse 80. Leukocytes, 12,500; neutrophils, 70 per cent; immature forms, 13. Slight infection; good resistance.

19. A case diagnosed clinically as multiple sinusitis. On September 22—leukocytes, 6,500; neutrophils, 32 per cent; immature forms, 5; eosinophils, 2 per cent. On October 10—leukocytes, 6,500; neutrophils, 55 per cent; immature forms, 3; eosinophils, 3 per cent. The blood picture shows no evidence of acute infection. The rather high percentages of lymphocytes in the two counts, 50 per cent and 36 per cent respectively, may indicate some chronic inflammatory, but with normal numbers of immature forms, it is apparently very slight.

20. Multiple sinusitis. September 17—temperature 102°F.; leukocytes, 5,400; neutrophils, 56 per cent; immature forms, 39. September 18—temperature 102°F.; leukocytes, 8,600; neutrophils, 63 per cent; immature forms, 53. September 22—



GIBSON CHART.

BRONCHO- PNEUMONIA. (Case 23).

temperature 99.8; leukocytes, 12,600; neutrophils, 60 per cent; immature forms, 38; eosinophils, 3 per cent. September 29—temperature 99.2°F; leukocytes, 11,900; neutrophils, 54 per cent; immature forms, 36. Temperature was normal on October 2. Moderately severe infection, but good resistance. A chronic condition had probably been present for some time before admission to the hospital.

21. Erysipelas. Temperature 102°F.; pulse, 110. Leukocytes, 26,000; neutrophils, 93 per cent; immature forms, 57. Recovered in five days under serum treatment. Severe infection and rather poor resistance.

22. Broncho-pneumonia. January 9—temperature 99.8°F.; leukocytes, 17,200; neutrophils, 83 per cent; immature forms, 52; eosinophils, 1 per cent. January 10—temperature 102.2° to 104°F.; leukocytes, 9,500; neutrophils, 90 per cent; immature forms, 83; January 13—temperature 99.6° F. to 102.6°F.; leukocytes, 24,200; neutrophils, 96 per cent; immature forms, 86. Severe infection with poor resistance. Died January 19.

23. Broncho-pneumonia. November 7—temperature 103°F.; leukocytes, 40,000; neutrophils, 93 per cent; immature forms, 69. November 8—temperature 103°F.; leukocytes, 37,600; neutrophils, 87 per cent; immature forms, 68. Severe infection but good resistance. Temperature was normal November 11.

SUMMARY.

A brief description of the leuckoytes and their origin is given.

Leukocytosis in acute conditions depends almost entirely upon an increase in the neutrophilic granular cells. At the same time there is usually a reduction in eosinophils.

The appearance of immature neutrophilic cells in the blood stream is taken as an indication of active infection, the activity being in proportion to the number and degree of immaturity of such cells.

The Schilling index brings out the significance of the immature neutrophils.

In infectious and inflammatory conditions, a comparison of the percentage of neutrophilic cells with the total leukocyte count yields more information than either alone.

By means of the Gibson chart, the relation of severity of infection to the patient's power of resistance is shown graphically.

By the application of the Schilling index and the Gibson chart, the surgeon can obtain much valuable information as to indication for operation, time of operation, and prognosis in operative cases.

The blood findings in a number of clinical cases are shown graphically.

BIBLIOGRAPHY.

(1) Piney, A.: Recent Advances in Haematology. 2nd Ed., P. 117; P. Blakiston's Son & Co., Philadelphia, 1928.

(2) Todd, J. C. and Sanford, A. H.: Clinical Diagnosis by Laboratory Methods, 6th Ed., PP. 291-293; W. B. Saunders Co., 1927.

THE HOME TREATMENT OF DIABETES.*

SEALE HARRIS, M. D.

BIRMINGHAM, ALA.

The many invitations that I have received during the past twenty years to address the Mississippi State Medical Association have been most gratifying to my professonal pride; because in no state have I friends for whom I have greater respect and esteem, and no state association has higher standards in its scientific programs than has your great medical organization. It is particularly pleasing to be with you on this occasion because Dr. Dearman, the Chairman of your Section on Medicine, has assigned to me perhaps the most important and the most practical subject that I have ever been called upon to discuss, i. e. "The Home Treatment of Diabetes."

It is estimated that one person in every 100 of population in the United States has diabetes, and probably 95 per cent of diabetics are treated at home by general practitioners. Therefore their diets must be prepared by mothers and housekeepers,

*Read before the General Meeting, at the Sixty-second Annual Session of the Mississippi State Medical Association, Gulfport, May 16, 1929.

many of whom cannot, or will not, learn how to weigh and measure food in grams and calories. Knowing the need for simplified methods of treating diabetes it has been a pleasant task to endeavor to construct a series of diets, expressed in household terms, which approximate the nutritional needs and the carbohydrate limitations of the average case of diabetes; and it will be most gratifying if these efforts prove helpful to the general practitioner and his diabetic patients.

SIMPLIFIED DIETS.

Since the discovery of insulin, which placed the treatment of diabetes on an accurate, scientific basis, many clinicians have been working on the problem of preparing simplified diets for diabetics, who, either have not the education to learn the arithmetic of diabetes, or who would not take the time to master the simple methods of calculating food values. Some of you may recall that at the meeting of the Southern Medical Association in Washington, in November, 1923, I presented a paper, "The Use of Insulin by the General Practitioner," in which was outlined a series of simplified diets for diabetics, arranged according to weight and age. We have been gratified to learn from many physicians that they have used successfully those diets in treating their diabetics at home.

Since we have found from experience that it is practicable in beginning the treatment of the uncomplicated cases of diabetes in adults to use one diet with which to test the patient's carbohydrate tolerance. This diet may be added to, or subtracted from, until daily menus have been worked out which will provide sufficient food for the diabetic to eat to satiety, maintain his normal weight, and carry on with ease and efficiency the duties of his vocation.

Of course it is best to treat diabetes scientifically, but since that is not always possible, we have followed the rule which was learned during ten years of general practice; i. e. that where the physician cannot do what is best for his patient, then do the next best thing, and above all things, apply common sense in the management of disease. If the diabetic has to be treated at home, even if he is intelligent enough to learn food values, in beginning his treatment it is usually best not to confuse him by discussing grams and calories but prescribe a diet selected largely from food that is prepared for the rest of the family.

It is important in the very beginning of treatment to impress upon the patient that it is not difficult to prepare the food for a diabetic, nor it is a hardship to live on such a diet; because he can have plenty to eat of a variety of foods, and if he will adhere to his diet diabetes will not shorten his life or interfere with his usefulness or happiness. The fact is that the careful diabetic will live longer than other members of his family who have not been taught to live on a low carbohydrate, well balanced diet.

Some of the most brilliant results that we have seen in the treatment of diabetes have been among very poor patients who could scarcely read, or write, but who possessed what many educated people lack, i. e. common sense, and the will to do right. They were placed on diets, containing given quantities of simple foods expressed in household terms, which they carried out in their homes. They were not confused with trying to work with the metric system, and they were not given the fancy, patented diabetic foods that are expensive and often misleading in their carbohydrate content. They were taught simple and practical methods of how to live at home and have diabetes.

COOPERATION OF PATIENT NECESSARY

If the diabetic is among the 30 or 40 per cent who will have to use insulin he should be taught that it is even more necessary for him to adhere to his diet

than if he had a mild case of diabetes. Above all things the diabetic should be made to realize the seriousness of his disease, and impressed with the fact that if he is not willing to follow the diet prescribed by his physician, and remain under his care, even after he has learned the arithmetic and chemistry of diabetes, he surely will suffer the consequences of his dietetics sins. The paths of the careless, self-satisfied and self-indulgent diabetics who sometimes think they know more than their physicians, like the paths of glory, "lead but to the grave."

In order to secure the full cooperation of the diabetic while keeping him under observation until his optimal diet can be worked out, it is necessary to explain to him that the only method of finding out the amount of food he can take and keep his urine sugar free is by trying out a test diet, increasing or decreasing the food, and using insulin if necessary, until the diet suited to his particular nutritional needs has been determined.

TRIAL DIET.

If the adult diabetic has no diacetic acid in his urine, if he can take solid food, and if there are no complications he is given the following trial diet, which may be increased or decreased until a diet is worked out on which he may live comfortably and keep his urine sugar free.

Breakfast: 1 raw fruit; ½ small grapefruit; ½ small orange; or medium size peach; or ¼ small cantaloupe. 8 tablespoonsful cream. 1 egg, cooked any way except fried, 1 pat butter on egg while hot. 3 slices bacon (crisp). 1 diabetic bran biscuit and 1 pat butter.

Dinner: I cup broth, bouillon, clear or strained vegetable soup. 1 Uneeda biscuit (cracker). Medium serving of lean meat, ½x3x4 inches (trim off fat); steak or roast beef; or ham, or roast pork; or fish; or ½ small, or ¼ large chicken; or 1 quail; or ¼ rabbit or ¼ squirrel. Three rounded tablespoonfuls cooked vegetables; turnip greens; or collard greens; or string beans; or asparagus; or okra; or cauliflower; or cabbage; or spinach; or 2 tablespoonfuls squash or beets; or carrots; or onions; or turnip roots. 1 pat butter on vegetables while hot. I diabetic bran biscuit.* 1 pat butter. Dessert: ½ baked apple; or 1 peach (sliced), or 2 tablespoonful strawberries, or blackberries, with 4 tablespoonsful cream (cream may be whipped). No sugar. No canned fruits or dried fruits.

Supper: 1 cup soup, strained, as for dinner. 1 Uneeda biscuit (cracker). American or Swiss cheese (½x2x3 in.) or 4 tablespoonsful home-made cottage cheese; or meat as for dinner. 4 tablespoonsful raw vegetable; celery, lettuce, cold slaw, or 1 large, or 2 small tomatoes; or 1 tablespoonsful Waldorf salad. Diabetic mayonnaise.** 1 Diabetic bran biscuit.* 2 pats butter. Egg and cream custard.*** Dessert: ½ small orange; or ½ small grapefruit; or 1 small or ½ large peach; or ¼ small cantaloupe; or small piece (about the size of a small orange) of heart of watermelon.

First Raise in Diet. The diabetic is kept on the trial diet for 3 days and if his urine becomes sugar free he is given a raise in

**Recipe for Bran Biscuits.* Use regular feed bran. Put 6 or 8 cups in sack and let water run through it, or stirring bran until water is clear. This usually requires an hour or more. Let this dry, keeping it on hand for biscuits. Pillsbury bran may be used without washing. Mix 2 cups washed brand, or Pillsbury bran, with one tablespoonbul mineral oil. Put one tablespoonful of agar-agar in 1 cup of water, heat to boiling point. Mix bran and dry ingredients with agar in water —make biscuits about 1 inch thick. Grease muffin tins with mineral oil—cook in moderately hot oven about 40 minutes.

***Recipe for Diabetic Mayonnaise* I egg yolk, 1 tablespoon lemon juice, ½ cup mineral oil. Place egg yolk and oil on ice. Beat yolk and add oil drop by drop, stirring constantly. This makes 5 servings of dressing.

****Recipe for Eggs and Cream Custard.* 1 egg and 3 or 4 tablespoonsful cream. Beat eggs thoroughly, add cream and mix well. Pour in container and bake in a slow oven.

the above diet as follows: Add to breakfast two teaspoonful oatmeal; two ounces (4 tablespoonsful) cream. Add to dinner three tablespoonsful cooked green vegetables. Add to supper one tablespoonful raw vegetables and 1 pat butter.

Second Raise in Diet. If the diabetic continues sugar free for 3 more days on the trial diet plus the first raise, he is given the second raise as follows: Add to breakfast two tablespoonsful oatmeal, after cooking. Add to dinner 2 oz. (4 tablespoonsful) cream, and 1 pat butter.

Third Raise. If the patient's urine continues sugar free after the two raises plus the trial diet he is given a third raise of 1 or 2 extra slices of bacon for breakfast; and an increase in the quantity of cooked green vegetables for dinner by 1 or 2 tablespoonsful. Likewise the amount of meat, fish or fowl may be increased by one-fourth or one-half for dinner or supper.

If the patient's urine remains sugar free on the trial diet plus two or three raises he will be getting about the optimal diet for the average adult, and he is then kept on that diet indefinitely, until there are indications for increasing, or decreasing his food.

Weighing Food in Grams. The trial diet and the raises when measured in tablespoons and ounces allow a considerable percentage of error, but since most of the foods used in these diets belong to the 3 per cent, 5 per cent and 10 per cent vegetables and fruits unless the error is very large it will make little difference in the total carbohydrate content of the three daily meals. It should be understood by the physician and his diabetic patient that the household measurements are not to be compared in accuracy with weighing and measuring food in grams; and the trial diet with quantities prescribed in tablespoonsful is not to be used except for a few days, with the patients who can learn the metric system. The trial diets and the raises were prepared for use by diabetics who cannot buy the gram scales, or who have not sufficient education to learn to weigh and measure their food in grams. We have found it practicable to teach even the most unlearned diabetics to weigh their food in grams, with diets that have been worked out for them. The effort to teach food values to many diabetics only confuses them. In such cases nothing is said to them about carbohydrates, proteins and fats, and the word calorie is never mentioned to them. They are persuaded to spend 10 dollars for a Chatillon, or Hanson gram scales, on which with movable dial, they soon learn to weigh their food. The trial diet with raises worked out in grams is given them; and with very little instruction they learn to weigh out their three daily meals. Indeed it often is best to show the intelligent diabetic how to weigh his food in grams before the effort is made to teach him the metric system, and the carbohydrate, protein and fat percentages, and caloric values of various foods. The following trial diet worked out in grams is useful in such cases:

Breakfast: 1 raw fruit: 150 gms. (½ small) grapefruit; 75 gms. (½ small) orange; 75 gms. peach (without seed); 75 gms. Cantaloupe or muskmelon (edible portion). 60 grams (4 tablespoonsful or 2 ounces) cream. 1 egg. 15 gms. bacon. 1 diabetic bran biscuit. 20 gms. butter (½ on egg).

Dinner: Soup; clear broth, bouillon or strained vegetable soup. 1 Uneeda biscuit (cracker). Meat: 50 grams lean beef; or steak; or ham; or chicken; or fish; or game. Vegetables: 100 grams (after cooking) turnip greens or collard greens; or spinach or cabbage; or asparagus; or okra; or 50 grams squash; or beets; or string beans; or carrots; or onions; or turnips. 1 diabetic brain biscuit. 20 grams butter (½ on vegetables while hot). Dessert: 50 grams baked apple; or 75 grams peach; or 50 grams pear; or 75 grams strawberries; or 75 grams blackberries; or 75 grams dewberries; or 50 grams rasberries; no sugar.

No dried fruits or canned fruits or canned berries. 60 grams cream.

Supper: Soup: 1 cup, strained as for dinner. 1 Uneeda biscuit (cracker). Meat Substitute: 40 grams American or Swiss cheese; or 50 grams cottage cheese; or 60 grams English (green) peas; or 60 grams lima or "butter" beans; or, if meat is preferred, 50 grams as for dinner. Raw vegetable: 30 grams lettuce; or 60 grams cold slaw; or 60 grams tomato; or 75 grams celery; or 40 grams Waldorf salad. Diabetic mayonnaise. If raw vegetables not available may use cooked vegetables as for dinner. 1 diabetic brain biscuit. 20 grams butter. Dessert: Diabetic egg and cream custard; or 150 grams (½ small) grapefruit; or 75 grams orange; or 75 grams peach; or 75 grams cantaloupe; or 75-100 grams watermelon (edible portion).

First Raise in Grams. If the diabetic's urine is sugar free after one, two or three days, on the trial diet weighed in grams, the first raise is made as follows: Add to breakfast 40 grams of oatmeal (after cooking). For dinner add 50 grams of green vegetables, making 150 grams of vegetables.

Second Raise in Grams. If the diabetic's urine remains sugar free or becomes so in one, two or three days on the trial diet plus the first raise, make the second raise consisting of 40 grams more of cooked oatmeal, making 80 grams of oatmeal for breakfast. Also add 60 grams cream, making 120 grams (4 ounces), for breakfast. 60 grams cream and 10 grams butter may be added to dinner.

Third Raise. If the diabetic's urine is sugar free, in one, two, or three days after the first two raises 10 grams (2 slices) of bacon are added to breakfast making 25 grams bacon for breakfast; and 50 grams more of green vegetables are added to dinner or supper. Likewise the amount of meat, fish or fowl may be increased by 25-50 grams.

Examining the Urine at Home. One of the first lessons that the diabetic should learn, whether he is treated at home or in a hospital, is to examine his own urine for sugar and diacetic acid. One or two test tubes, a few ounces of Benedict's solution, a small bottle of ferric chloride, and a medicine dropper will not cost the patient over a dollar, and that is all he needs to keep informed as to whether or not he is eating too much glucose forming food and also as to whether or not he is metabolizing his fats.

To test the urine for sugar add 8 drops of urine to a teaspoonful and a half of Benedict's solution in a test tube, shake and place it in a cup of boiling water on the kitchen stove, and let it simmer for five minutes. If a heavy greenish precipitate forms it means a trace of sugar, if a yellowish sediment is found a moderate amount of sugar is present, and if the precipitate is red it shows that the urine is heavy with sugar.

For several weeks after beginning the treatment the patient or some member of his family should examine two or three specimens a day until he learns the diet he can follow and keep his urine sugar free; and he should examine his urine frequently as long as he lives. If the diabetic is tempted to eat sweets; or too much bread or other starchy foods; or if he eats too much meat, 58 per cent of which is metabolized as glucose, and then when he examines his urine and finds it loaded with sugar, he will not be so apt to repeat his indiscretion in eating.

The test for diacetic acid is just as simple. To about 2 teaspoonsful of fresh urine add 6 or 8 drops of liquor ferri chloridi, U.S.P. If the urine turns reddish a small amount of diacetic acid is present; but if it turns a deep Burgundy red, it shows a tendency to acidosis, an indication for the diabetic to reduce his fats or cut them out altogether, until there is no diacetic acid in his urine. It is better, however, to teach the diabetic to send for, or report to, his doctor when he finds the heavy red

color on adding the iron to his urine. The diabetic should know that if he takes aspirin the ferric chloride test for diacetic acid will be positive.

When and How to Use Insulin. If after three days on the trial diet there is still sugar in the diabetic's urine, he is given from 3 to 5 units of insulin 20 minutes before eating two or three times a day; and if in 24 hours his urine still contains sugar he is given from 6 to 10 units of insulin three times a day. If in another 24 hours the sugar has not disappeared from the urine, 15 units are given three times a day. In the severe cases 20, or even 25 or more units may have to be given three times a day before the patient's urine becomes sugar free, but in the majority of cases 5 or 10 units two or three times a day will be sufficient. When the patient's urine becomes sugar free on this diet from the use of insulin, if necessary to gain weight or to maintain bodily strength and vigor the diet is raised as above outlined and if the sugar returns in the urine insulin dosage is increased until the urine is free from sugar. Then the second raise may be made, and if the sugar reappears in the urine the insulin is increased by 3 to 5 units before each meal, each day until his urine becomes sugar free.

The third and fourth raises may be made in the same way, increasing the insulin slowly until the urine is sugar free. After the patient's maintenance diet and his insulin dosage have been worked out as above described, he is kept on it, with the same dose of insulin for several weeks, the effort being to keep the urine sugar free, without insulin reactions. If the insulin reaction occurs, the insulin is reduced or the food increased.

Discontinuing the Insulin. In many cases of diabetes after a few weeks rest to the pancreas by dieting and by the use of insulin, the patient's carbohydrate tolerance is increased. In such cases the insulin dose is gradually decreased and if the sugar does not reappear in the urine the insulin may be discontinued permanently. When the insulin is discontinued the patient should be made to realize that he should never over eat, that a food debauch may break his carbohydrate tolerance and he will then have to use insulin again.

Insulin is not habit forming, but it should not be left off suddenly without at the same time reducing the diet in proportion to the amount of insulin used. The patient taking insulin should keep on hand an adequate supply, but if he cannot get insulin he should reduce his food by one-half, or even two-thirds, and leave off the fats altogether, because the principal danger from leaving off insulin suddenly is acidosis, or even coma. It is not leaving off the insulin that is harmful, but continuing the extra food that he could metabolize by using it.

The patient using insulin should be made to understand that if for any reason his food is cut down his insulin dose should be reduced in proportion. If he fasts a day he must use no insulin without explicit directions from his physician; and if diarrhea develops the dose of insulin should be reduced because food may pass through the intestines without digestion and the diabetic with diarrhea may have a low blood sugar, which, with the addition of insulin, may produce an insulin reaction. A number of clinicians have reported hypoglycemic reactions from the use of even small doses of insulin with diabetics who have diarrhea.

For the above statement of facts the idea that once a person begins the use of insulin it is dangerous or harmful to leave it off is proved to be erroneous. It is true that the severe diabetic may have to continue insulin for the rest of his life, but without insulin

he could not live long. With insulin and the proper diet there is no reason for diabetes to interfere with the health, or efficiency of the most severe case of diabetes. Insulin is therefore a great boon to diabetics and thousands are living today who would have been dead without it.

While insulin has saved many lives it is a distressing fact that the diabetic death rate has increased since Banting's great discovery. This perhaps is due to the fact that many diabetics have received the impression that they can eat what they want, provided they use insulin; and no doubt insulin is being used injudiciously by inexperienced physicians. The management of the uncomplicated case of diabetes at home is comparatively simple; but it must be admitted that in dealing with diabetic children and with the complications of diabetes, such as acidosis, gangrene, the infections, and eye manifestations, a larger proportion will be saved if treated in hospitals under the care of clinicians who have had ample experience with the disease.

DISCUSSION.

Dr. Charles LeBarron (Gulfport): I was unfortunate in missing the first part of Dr. Harris' paper which I regret very much. I am glad to see the concise and even way in which Dr. Harris has presented a diet which may be used in the home.

We find, however, with all the various charts we have—carbohydrates, proteins, fats and calories—we have great difficulty in getting our patients to carry out this proper diet. We have three cases of people with whom we have to deal. First, we have the ignorant class of people upon whom it is absolutely impossible to impress the necessity of carrying out their diet and weighing their food and examining their urine regularly.

We have a second class of people, intelligent people, who are willing and active in their cooperation. In these we get the very best results.

We have a third class of people who seem to think they know more than the physician or dietitian, and as a result we have a woeful failure in these cases. When they find they are feeling pretty well and when their urine is practically sugar free, they feel as though they can get off their diet and do as they see fit.

Another great trouble I find with my patients is that they believe that insulin is a specific. They think if they take insulin they can eat what they want, drink what they want, and do what they want. I want to register a protest now against this idea which is prevalent among the people generally that insulin is an absolute specific for the treatment of this disease. I have had cases which have been obliged to continue the use of insulin for years and years and will probably continue it until the end of their days.

There is very little difficulty in following this diet because we have so many charts that we can give to the patients, and with ordinary intelligence they can figure out their diet without any difficulty.

I have enjoyed Dr. Harris' paper very much.

Dr. Joe E. Green (Richton): I want to thank Dr. Harris for bringing us this practical discussion. If there is anything I believe in, it is horse sense in everything and getting away from things that are technical and getting down to the common-sense things that we can use every day.

Dr. Harris has certainly brought us that this morning, very scientific but still simple as an old shoe which has seven holes in it. This is the sort of thing we want to carry back home. It will be helpful to us doctors. I feel sure that we are all mighty glad that Dr. Harris had a chance to present the paper, even though he got here late.

Dr. Seale Harris (closing): I have nothing to add in closing the discussion, except to say that Dr. Dearman is responsible for the title of this paper on the home treatment of diabetes. We have simplified the treatment so that the patient can carry it to his home. I feel very happy in being able to present this paper, and I want to thank you for your kind consideration and attention.

THE SIGNIFICANCE AND THE LIMITATIONS OF THE RADIOGRAM IN THE DIAGNOSIS OF ORAL SEPSIS.*

SIDNEY L. TIBLIER, D. D. S.,

NEW ORLEANS.

I deeply appreciate the honor and privilege to address you. As an associate member I have been very much interested in your deliberations for they have some bearing on my chosen field just as I believe the subject I have chosen to speak on has a bearing on all phases of medical science.

The subject of oral sepsis is a wide one and it is impossible in the time allotted to go into all phases of it.

As a dentist I feel that my remarks should be limited to the pathology of the teeth, and their supporting and investing structures.

I believe I can say without fear of contradiction that the radiogram can be used to the greatest advantage in tooth and jaw work for in this region we have structures which are all definitely radiopaque and in varying degrees; making it possible to determine many pathological changes not so easily demonstrated in soft tissues.

The tooth alone by virtue of its three distinct calcified tissues, histologically different, show up differently in the radiogram, so that deviations from normal can easily be demonstrated.

For instance, destruction of enamel by decay, building up of secondary dentin, resorption or hypercementosis of the cementum. And then, when we consider the nature of the surrounding cancellous and compact bone which shows up in clear contrast to the teeth it is possible to note very easily bony changes in the direction of rarefaction or condensation or complete destruction as in necrosis.

These considerations form then the basis of the significance of the oral radiogram.

Sadly, however, we are confronted with the fact that because so many things of diagnostic value can be told even from a poor radiogram that many of the finer details for a complete diagnosis are lost.

For instance, the incipiency of peridental disease as shown by a break in the lamina dura surrounding the tooth.

Or again, the pathology at the end of a root may be expressed in terms of slight resorption or roughening and this is lost sight of in the poor radiogram, and we could go on ennumerating a great number of examples.

Now with all the significant features of the radiogram, even a poor one, let us consider the limitations and the means we have at our disposal to make complete diagnosis, by making up for these deficiencies in the radiogram.

First, the radiogram gives us little information regarding the etiological factors of the evident pathology found in the film. These factors are either systemic or local and must be learned in order to make a complete diagnosis.

Secondly, the radiogram shows the teeth in their mesiodistal relationship and tells us nothing of pathology which might exist labially or buccally and lingually of the teeth.

Third, the radiogram does not show an incipient gingivitis for no bone changes have as yet occured. The case might be on the road to an extensive pyorrhea which might be prevented by determining the etiological factors concerned in the gingivitis and eliminating them.

Fourth, the radiogram alone is not always conclusive about the relationship of infected teeth to the maxillary sinus.

*Read before the Orleans Parish Medical Society, June 24, 1929.

Fifth, the radiogram tells us nothing of the bacterial nature of the infection. We guess very often but that is not scientific.

Sixth, it is impossible to tell merely from the radiogram whether an area of bone destruction or loss represents infection without the history and clinical examination at hand. We will show that in a slide.

Seventh, can we definitely say that any area is acting as a focus or not from the radiogram? I think not. However I am of the opinion that when found they should be removed and not wait for a heart lesion, or kidney lesion, etc. to develop. Better prevent than attempt cures and I would underscore the word attempt for very often no seeming good results from the removal of foci for the damage is done. Removal of foci are always productive of good, but the good is oftentimes only expressed by an inhibition of further damage and an increase in resistance.

We could go on with innumerable other limitations of the oral radiogram but they will be brought out in considering the slides.

I would make the plea that the radiogram be taken at its face value and the positive assertion that it should be supplemented by the following procedures at least if a fairly complete and an accurate mouth diagnosis is desired.

History, dietary habits, mouth mirror and explorer examination, blunt probe examination of the gingival crevices of all teeth for pockets, transillumination of all teeth and gums and of the sinuses, vitality test of all teeth and relative vitality as compared to normal; study models of the case to determine the local etiological factors often expressed by malocclusion and trauma and lack of function, differential diagnosis especially in tumor formations.

These procedures should be routine for all cases and is the part that the dentist is responsible for.

The blood examination, urinalysis, serological tests and differential diagnosis of those conditions often occuring in the mouth which are manifestations of systemic disease, the physician is responsible for.

As physicians you are vitally interested in eliminating foci of infection and since these foci are some times obscure it behooves you to have thorough diagnoses made.

Many teeth have been uselessly sacrificed but I believe many infected teeth with obscure pathology have been allowed to remain because of slip shod methods of diagnosis or a total dependence on the radiogram for diagnosis.

A full mouth roentgen-ray examination together with the procedures outlined above is routine in my practice even for patients in good health and I think is the foundation of good sound preventive dentistry, health dentistry and should be practiced by all dentists, and I feel that when I discover and eliminate an area of infection which is liable at any time to begin its slow insidious work of affecting other vital organs perhaps to the point of irreparable damage, I have done something worth while.

DISCUSSION.

Dr. Lucien A. Fortier (New Orleans): I enjoyed Dr. Tiblier's paper very much indeed and think he has thoroughly covered the subject.

From the title, I feared he estimated roentgen-rays as being of little worth in diagnosing peridental infections, but after seeing his films and hearing his demonstrations, I feel much relieved to learn that they are of value.

We, who do general radiographic work, realize that the dentist has to see the case; has to make a thorough examination; and must co-operate with us in order to do the most good to the patient. In the treatment of medical cases, the real sick patient requires that every possible focus of infection be removed, i. e.: in heart disease, rheumatic conditions, or any other condition in which a focus of infection might be the cause of the disease. Prophylaxis is a step forward in cases where there is definite evidence of peridental infection. I believe foci of infection ought to be removed.

I wish to again state that I enjoyed the doctor's paper very much.

Dr. Homer Dupuy (New Orleans): Dr. Tiblier's first contribution before this society is worthy of high commendation. The radiograms, unusually clear in outlines, with h's comments are very instructive. They prove his contention, that too much dependence is not to be accorded the roentgen-ray in dental pathology. I am convinced that this attitude applies to the whole skull. Rightly does he say that the history and clinical examination must be carefully considered before reaching a conclusion. A roentgenogram when taken by a good technician is of great help, but, it is never infallible or final. Relative to the maxillary sinus, or antrum, I know he is right. Radiograms do show, in certain subjects, teeth roots projecting into the antral floor. But, it requires clinical proof to accuse a tooth as an offending factor. On the other hand we have instances in which teeth with obscure pathology are allowed to remain in antral floor, when they are causing damage to the sinus. And this resulted for lack of clinical examination when too much reliance was placed on the inconclusive evidence of radiograms. And then we have the all-important question, is the antrum playing the role of a reservoir for pus accumulations from the upper sinuses? I have proven that teeth projecting into the antrum may thus become infected secondarily. Roentgen-ray will only give us the half truth in such conditions. Nothing but the most refined clinical examinations will clear up such a problem.

Dr. W. A. Lurie (New Orleans): We must compliment Dr. Tiblier on his presentation of so comprehensive a paper on so broad a subject, and in so short a time. I agree with much of what he has said of the fallacies in the taking and in the reading of the dental roentgenogram. It has been my practice to classify the shadows as seen in these pictures into three grades, identified as grades I, II and III depending upon the density of the tissue or body producing the shadow. By such division of shadow class one may better understand the type of condition one is dealing with, and the direct relation it bears to some tissue type. Relative to the seriousness of focal infection there is no discussion. Some of the so-called failures of relief, however which are recorded after the removal of the oral focus of infection are due as the doctor intimates, to the development of an active secondary focus, un-recognized or untreated, and very often to incomplete removal of the original focus, as is often noted in the dental radiogram.

Dr. Tiblier's paper is on a vital subject and I believe we should devote more time to discussions of this character.

Dr. Sidney L. Tiblier (closing): I certainly appreciate the kind way in which the discussions went along about the subject that I chose and am glad that those who discussed it were practically in accord with what I had to say.

However, to emphasize one point I brought out in this work about health dentistry, I want to add this: that if every dentist would follow what I term as routine in the handling of these cases, many of these severe infections could be prevented. We are getting away from the old-time dentistry of waiting for cavities to become large before filling them and of telling a patient with a history of tooth trouble to wait for further developments and to report back in a month or two. There is no telling what will happen in the interim. The pulp may become involved and even the bony structures beyond affected. Some teeth go to wreck in a very short time. By routine examination, using the radiogram for its real value in determining pathology, we can prevent a lot of trouble. Trace the condition down to the etiological factor. How can a positive diagnosis be made unless the dentist knows something of the cause? You do not find physicians treating diphtheria, for example, without getting cultures and smears made. Some men will not give antitoxin until their diagnosis is confirmed by laboratory study. Why? Because they insist on knowing the causative agent. A good many dental conditions show up in the roentgenogram, and in some instances by not knowing the etiological factor and waiting for further developments a tooth is lost and the only treatment left is extraction. A natural tooth is better than any restoration, yet I agree with Dr. Fortier that a tooth that is a source of infection should be removed. In cases of suspicious teeth in cases of systemic disease the co-operation of the medical man, the roentgenologist and the dentist will insure a correct diagnosis and good work.

I am certainly very pleased and feel honored to have addressed you tonight.

Thank you.

NOTE: This paper was supplemented by lantern slides.

SPINAL ANALGESIA IN ABDOMINAL SURGERY.*

ANDRE B. CARNEY, M. D.,

CLARKSDALE, MISS.

During recent years the question of anesthesia has played a major role in all surgical discussion, and during the past year it has become unusual to look through any one of the current surgical magazines without some reference being made to spinal anaesthesia.

The older custom of a general anaesthetic to most patients is to a certain extent in different sections of the country being somewhat displaced by the more technical, though very satisfactory, spinal analgesia. This procedure, while now apparently coming into its own, has enjoyed previous spasmodic eras of popularity since 1885, though due to a former definite and distinct misunderstanding of the exact physiological and chemical reaction in the spinal cord the procedure was employed with a certain degree of fear and trepidation, even by those most familiar with its effects. Notwithstanding the cautiousness employed, certain occasional unfortunate results have reacted to its present disadvantage. Due to recent works by Labat, Koster, and Kassman (Surgery, Gynecology and Obstetrics, November, 1929) and others, we known the exact nature of the reaction occurring and can use certain definite procedures to prevent the untold results.

A brief historical review advises us that spinal anaesthesia was first suggested by Corning in 1885; but it is to Bier that credit must be given for endowing surgery with the method. In 1898 Bier started with cocain as an anaesthetic agent, and obtained complete anaesthesia of the lower half of the body, although in one patient the analgesia extended as high as the scalp; but the after effects, chiefly giddiness, severe headache, vomiting, and syncope, were most disturbing. Being under the impression that these symptoms were due to loss of cerebrospinal fluid Bier kept his patients in bed for a long time. This did not help to relieve the condition so he dropped the method as being too dangerous, and decided to await the discovery of a less toxic drug. The method was, however, continued by Tuffier in France, despite the considerable opposition and notwithstanding the fact that it had been practically given up in Germany.

In 1891 Giesel isolated tropacocain from the leaves of the Java Coca plant. The alkaloid was synthetically prepared by Liebermann in 1892, and has since enjoyed great favor in Germany. Its toxicity is half that of cocain. It was only in 1904, when Fourneau discovered stovain, that spinal anaesthesia was again brought before the profession. Novocain was introduced the same year by Einhorn. It was found much less toxic than any of the anaesthetic agents known at the time. Its combination with adrenalin gave it a high practical value in the method of local anaesthesia, for which it is still considered as the ideal anaesthetic agent; but when injected with the dura it was said to be less active than the other drugs. After a few trials with novocain, the pioneers of spinal anaesthesia went back to stovain. Some surgeons used tropococain; the minority reinjected cocain in the weaker doses of purer solutions. The ill-effects of the drugs employed and the occasional fatalities due to the method form the subject matter of many reports. In the meanwhile careful experiments were made with novocain on the human being, and the clinical observations actually tend to show that novocain must be preferred to the other anaesthetic agents for the induction of spinal anaesthesia.

It is not the author's intention to discuss the reasons why novocainization is safer than the other methods of spinal anaesthesia, nor to dwell upon the merits of any individual technic. When dealing with a method which, for so many years, has met

*Read before Clarksdale and Six Counties Medical Society, November, 1929

with considerable opposition, especially on the part of those who had little or no experience of its principles, one should be cautioned against trying too many procedures and experimenting with various drugs. Fatalities are mostly due to lack of experience; their occurence is always detrimental to the method. Let those surgeons who are familiar with one method stick to it, if they consider that the advantages derived from it are the best; but those who are anxious to use spinal anaesthesia for the first time, or desirous of improving the technic they are actually using, will find in the following pages a handful of information gathered from personal experience.

In recent years, however, reasonably safe solutions of intra-spinal preparations have been placed on the market. These preparations in the hands of the average surgeon, exercising average care and precaution, can be, and are used as safely and efficiently as the several forms of general anaesthesia.

The preparation with which the majority of us are most familiar, I am sure, is that marketed under the name of spinocain. This preparation is put in sealed glass ampules ready for immediate use. By being so prepared the old danger of contamination is lessened. The preparation is marketed in two strengths though the weaker solution of approximately gr. IV of novocain to an ampule is the one most generally used.

Spinocain has a lighter specific gravity than spinal fluid which is an important factor in the form of anaesthesia and a factor which must be considered beforehand if unfortunate results are to be avoided.

This factor, as I shall show later is one of the most important points in securing a good spinal anaesthesia, or as it should correctly be called spinal analgesia.

The technic of spinal analgesia is relatively simple.

The patient is placed on the operating table in the prone position. He is then requested to turn on his side, the left side, if right surgery is to be performed, and the right side if left surgery is the case. There is no choice if a mid-line incision is to be made. The knees and hips are acutely flexed or as nearly so as possible. The shoulders and hips are in the vertical if ease of puncture is to be established. On examination of the spinal column wide interspaces are noted between the bodies of all vertebrae below the level of the lower thoracic. The site of choice for ease of puncture and good analgesia for all areas below the level of the umbilicus is the space between the fourth and fifth lumbar vertebrae, however, if surgery of the upper abdomen is being done a higher interspace is preferrable though not essential.

The technic of introducing the anaesthetic with the patient in the position described above is done with the same care as in preparing for the operation. The area of skin is painted with the desired strength of iodin, usually 3 per cent, though the ordinary full strength tincture is satisfactory. Sterile towels are applied to the surrounding adjacent parts in order to minimize the danger of introducing infection. With the gloved finger an indentation is made at the selected site of puncture. Into the skin and deeper tissues of this area is injected 1 cc of solution of novocain containing m 3 of adrenalin hydrochloride. This prevents puncture pain in the skin while the adrenalin aids in maintaining normal blood pressure following injection of the intra-spinal solution. The needle for the spinal puncture should be one with more or less blunt beveled edge. This is of decided advantage in not going through the canal as is frequently the case with the long-pointed, sharp needles. The needle is grasped by the syringe end and the shaft, not near the point, as this is unnecessary, and is only inviting infection. As the canal is punctured there is a peculiar feel that comes only with experience and similar to the feel occasioned by sticking a pin through a tightly drawn piece of rub-

ber. The stylet is slowly and carefully withdrawn, care being taken not to disturb the position of the needle.

The syringe loaded with the anaesthetic agent, and in such position that it can be picked up without the operator moving materially, is now attached to the needle. A small amount of spinal fluid is withdrawn to make sure that the needle is still in place. The anaesthetic is now very slowly injected and withdrawn. This procedure is repeated several times to insure complete diffusion of solution in the spinal fluid, and unless this is done the results will be very disappointing. The amount of diffusion necessary depends to a great extent on the site of the operation. If the work to be done is of the vaginal plastic or rectal work either once or twice will be sufficient.

If the procedure is for a hernia or appendix then three to four diffusions are necessary to insure good analgesia. For work on the gall bladder or stomach it is best to puncture the canal between the last thoracic and first lumbar and complete the analgesia with three of four diffusions. This hastens the effects as well as allows the operator a little more surgical time, however, a successful result for this area can be completed with puncture between fourth and fifth lumbar vertebrae, though the anaesthetic agent must be thoroughly diffused and patient held in horizontal or five degrees Trendelenburg for at least ten minutes.

After the solution is completely diffused in the canal the patient is turned flat on his back and the head of table is dropped to the desired degree of Trendelenburg. It is unsafe to operate with less than 5 degrees and safety factor is directly proportionate to the degree of Trendelenburg. Since this solution is lighter than spinal fluid the experience of the surgeon determines to a great extent the degree of Trendelenburg desired and the higher the operative fluid the less Trendelenburg in degrees can one satisfactorily employ. To attempt a high operation in a deep Trendelenburg position would necessitate a decrease in the time factor if good analgesia were to be maintained throughout.

The above agent properly administered is surgical for approximately one hour depending to some extent on the individual and the degree of toxicity present.

Following the administering of this form of solution the patient should at no time be allowed, within three hours, to become horizontal or sit up. After removal from the operating table, the patient is placed on the bed, the foot of which has previously been elevated to the desired degrees and allowed to remain in this position during the intervening length of time.

The danger in this form of analgesia is primarily in the fall of blood pressure resulting in cerebral anemia and not in the respiratory or cardiac paralysis as was once believed. Should the patient show signs of embarrassment the Trendelenburg position should be increased immediately and be maintained until the patient feels entirely comfortable. After this, if it is advisable to do so, the degree of Trendelenburg is very slowly decreased. Adrenalin m x may or may not be given, as the operator sees fit.

This form of analgesia is very desirable in many toxic cases where a prolonged general anaesthetic would be detrimental. The surgical shock in all cases is apparently greatly minimized thereby lessening in our toxic cases the necessary high mortality. As a general rule in all cases with blood pressure not less than 100, all other factors being equal I believe it affords the surgeon at present the safest and most reliable form of anaesthetic agent.

BACILLARY DYSENTERY IN LOUISIANA.

DANIEL N. SILVERMAN, M. D.,

NEW ORLEANS.

Attention is called to the occurrence of bacillary dysentery in this state and city, not because the writer has anything new to offer in the way of diagnosis or treatment, but as a reminder of the outcropping of acute cases in sporadic instances. Some of these cases are allowed to go undiagnosed and untreated until a state of chronic dysentery is developed. Others do not appear for supervision until several weeks to months have elapsed since the acute attack. Fortunately, according to some authors, there is a tendency for the acute cases to heal spontaneously while a few merge into a chronic condition. In a previous communication in the Medical Clinics of North America the writer reported a small series of cases of chronic bacillary dysentery, calling attention to the frequency of the disease, the fact that many are treated with one form or another of anti-amebic medication without cure. While amebiasis is sometimes associated, the coexistence of the two diseases is sometimes accountable for our failure to obtain complete cures of individuals who have had amebic dysentery and very adequate treatments for same.

In the Touro Infirmary for a period of ten years (1919 to 1929), there were a total of 25 cases of both acute and chronic bacillary dysentery. Out of this number, 8 were in babies and children between the ages of 3½ months and 4½ years. The children's cases were acute in 7 and chronic in 1. Three of them died within a few days of the onset, a mortality of 37½ per cent. Of the 17 cases in adults, 7 were acute and 10 chronic. There were no deaths. The geographical distribution of the twenty-five cases show the majority originating in New Orleans, namely, 15. The other cases were about equally distributed in north, southwest and south Louisiana with the exception of the two that came from Mississippi.

The present paper will deal principally with a discussion of cases in the acute form. With reference to the diagnosis, it is occasionally necessary to depend on the clinical findings for a very early diagnosis. The agglutination test may appear later and the bacilli are not infrequently missed. In each of my cases the agglutination test was strongly positive in a few days after the onset of symptoms. Whitmore states that the agglutination reaction is not of much value before the tenth day of the disease. On the other hand, some authorities have found agglutinins in the sera of patients as early as the third or fourth day of the disease, and some have found the agglutinins in the sera of half of their patients before the tenth day of the disease.

CASE REPORTS.

1. J. W. W., aged 53 years, a salesman, while traveling in North Louisiana, became suddenly ill with abdominal pain and bloody diarrhea on July 3, 1925. He was seen by me one week later. The examination showed a temperature of 103 3/5 degrees, pulse 115. The blood count showed a total white of 9,150 with 90 per cent neutrophils. The bloody stools presented numerous pus cells but no ameba. The proctoscopic examination revealed an extensively ulcerated rectum and sigmoid with negative smears from the ulcers. The cultures were negative for dysentery bacilli. The blood agglutination was positive against B. dysenteria of Flexner. The temperature subsided within four days of rest in bed and fluids only. The patient insisted on leaving the hospital. The bowel mucosa continued to present numerous ulcerations.

2. A colleague awakened during the night in August, 1927, with rather severe abdominal cramps, an intense diarrhea of blood and mucus. The temperature rose to 102° and for about ten days varied from 99.5° to 102°. A slight leukocytosis of about twelve thousand was present at the outset. Within three days the blood area was positive for agglutinins against B. Shiga, in dilution of 1-60. The sigmoid was very muchly injected and ulcerted. On absolute rest in bed, a liquid diet, the disease subsided in two weeks and there have been no symptoms of dysentery since.

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3. Dr. B., an interne in one of the local hospitals and a resident of New York, had been sick for one week with frequent stools and general weakness. His abdomen was generally tender, especially over the cecum. The stools, numbering 8 to 10 daily, were bloody and mucoid containing much pus but no ameba or other protozoa. The temperature went no higher than 101°. The leukocyte count was 12,000 with 82 per cent neutrophils. The rectal and sigmoid mucosa was extensively ulcerated. One day after admission the agglutination test was weakly positive for B. dysenteria of Flexner. The very next day, eight days after the onset, the test was strongly positive (complete agglutination at 1-60. No bacilli dysenteria were found in the stools.

SUMMARY.

The above cases serve to emphasize the occurrence of sporadic cases of acute bacillary dysentery in Louisiana. Most of these cases are due to the Flexner bacillus and the mortality in adults is practically nil.

DISCUSSION.

Dr. Charles W. Duval (New Orleans): It is a pleasure to discuss the paper of Dr. Silverman on bacillary dysentery, particularly because of my interest and close contact with the subject for so many years. There are a number of interesting things about bacillary dysentery which unfortunately Dr. Silverman did not touch upon, and I can only allude to since the time allowed me is limited. Prominent among these which I might mention are etiology transmission, character and location of the lesion, complications, cure and prevention.

I have often thought that here in New Orleans we frequently overlook bacillary dysentery infection, due perhaps to the fact that we are too prone in this particular clime to regard dysentery in the adult as always amebic. We should not forget that bacillary dysentery is not only common but often associated with amebic infection. I have seen quite a number of bacillary infections associated with amebic colitis. These cases do not satisfactorily clear up after the treatment for amebic dysentery because of the bacillary infection which persists as a chronic form of the malady. Since amebiasis not infrequently is complicated with a Shiga infection the recognition that a mixed infection exists is important from the standpoint of treatment. In these mixed infections the bacillary dysentery may persist long after the cure of the amebiasis. I believe the physician would do well to determine in all cases of amebic colitis whether or not there is bacillary invasion as well, which is done by testing the blood for specific agglutinins, in other words performing the agglutination reaction test against members of B. dysenteriae.

It is important to bear in mind that bacillary dysentery is caused by several variants of the dysentery bacillus species, and the two more common strains are known as "Shiga" and "Flexner" respectively. The Shiga type of organism is responsible for the epidemic form of the disease while the Flexner strain is rarely found in the epidemics and is the cause of sporadic cases of acute dysentery. Furthermore the "Shiga" type produces a soluble toxin while the "Flexner" is endotoxic in character. The recognition of this difference in the nature of the toxin is important in connection with serum-therapy. There is an effective antitoxin on the market for the cure of the "Shiga" infection and no immune serum of value for the "Flexner" type of the disease.

Dr. Silverman spoke of the agglutination reaction as of value in diagnosis. I would like to add that it has the same diagnostic value as the Widal in typhoid or paratyphoid fever. The specific agglutinin occurs quite early in the blood of patients suffering with acute bacillary dysentery and may be detected on the third or fourth day of the disease.

There is another form of treatment for bacillary dysentery which according to some in more efficacious than antidysenteric serum. I allude to the bacteriophage of D'Herelle. This author claims that in four to eight hours after the administration by mouth of the dysentery "phage" there is a marked subsidence of the symptoms; the bloody mucus stools which have been eight to ten a day drop off to two and three, and the case is on the way to convalesence within twenty-four hours. While D'Herelle considers the "phage" treatment of bacillary dysentery specific. Davison and others have not gotten the same results. Perhaps they did not give it a fair trial. Certainly the "phage" treatment is to be tried and I would commend it to Dr. Silverman and others interested in bacillary dysentery.

Dr. E. C. Faust (New Orleans): An early differential laboratory diagnosis between amebic and bacillary dysentery is extremely important. In amebic colitis the finding of the active motile Endamebe histolytica, with or without ingested red blood cells, in the freshly passed stool, or the precystic or cystic stage of this endameba, constitutes a specific diagnosis. However, it is frequently true that the specimen is cold, in which case the vegetative amebae would have disintegrated. A macroscopic examination on the part of an experienced internist or laboratory worker

usually makes an immed ate diagnosis possible and allows of treatment 24 hours or more before culture methods yield results. The cellular exudate in bacillary dysentery consists of a preponderance of pus cells with little or no feces, while that of amebic dysentery contains a larger amount of red cells and copious fecal material. It is particularly important to have this information where bacillary dysentery is suspected in order that the toxic symptoms may be relieved. It must be remembered, however, that in a certain percentage of cases bacillary infection is superimposed on amebic colitis, in which case attention should first be directly to reducing the former, leaving the latter for subsequent treatment.

INTESTINAL DIVERTICULOSIS.*

J. HOLMES SMITH, JR., M. D.,

NEW ORLEANS

Diverticula, occurring in the intestinal wall, have been recognized for many years, but until rather recently, with the exception of Meckel's diverticulum, they have been of more interest to the anatomist and to the pathologist, than to the clinician.

Diverticula are pouches in the intestinal wall, covered by one or all of the coats of the intestine and connected, directly, with the intestinal lumen.

Depending, mainly, upon their covering, diverticula are classed as either congenital (true) or acquired (false). Congenital diverticula possess all of the coats of the normal intestinal wall. Of this variety, Meckel's diverticulum is the best and practically the only example. Other than Meckel's diverticulum, congenital diverticula are so rare as to be almost negligable.

The present paper is devoted to a brief consideration of the so-called, acquired or false diverticula, with a presentation of several cases. These diverticula differ from the congenital type, in that they do not ordinarily possess a muscular coat. Nothnagel defines them as "herniform protrusions of the mucosa through the muscular coats; at the same time the serous lining is pushed out by these hernias so as to cover the diverticulum." This does not seem to be an invariable rule, as at times diverticula, apparently acquired, are found possessing all of the coats of the intestinal wall.

As to causation, many theories have been advanced, all of which would seem to play a part yet none of them entirely so. All seem agreed that advancing age, constipation and weakened intestinal walls predispose to diverticula. Especially would this seem to be so with the colon. While this may be largely true, it does not answer for those cases occurring in young subjects. One of the cases reported below was but twenty-two years of age.

Congenital defects undoubtedly play a part both in the small intestine and in the colon. Another factor, apparently of great importance in those portions of the intestine which have a long mesentery and permitting of great mobility, is traction. This naturally would concern the jejunum, ileum and transverse and sigmoid colon.

It is not unlikely, also, that ulcers in the intestinal wall may predispose to the formation of diverticula by producing what may be termed a point of least resistance. This would seem to hold particularly true for diverticula occurring in the duodenum.

While formerly little attention was paid to this type of diverticulum, the reason was, no doubt, to be found in the fact that their diagnosis can only be made, definitely, by radiologic, surgical or post mortem examination. There is no symptom or train of symptoms characteristic of this condition. Routine radiologic examination of the gastro-intestinal tract has resulted in the finding of numerous unsuspected diverticula, many of which, however, have no clinical significance and are merely coincidental findings. Diverticula may occur in any portion of the intestinal tract from the pylorus to the rectum. The most common location is in the large intestine, but diverticula are not infrequently found in the

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small intestine. The most common location in the small intestine seems to be the duodenum.

Case[2] reports 6,847 complete barium meal studies, followed by suplementary barium enemas, with the following results:

"Evidence of duodenal diverticulosis in 85 cases. Evidence of jejunal diverticulosis in 4 cases. Evidence of diverticulosis in both jejunum and ileum 1 case. Colonic diverticulosis in 138 cases."

Because of their greater frequency, colonic diverticula are probably the more important, clinically, but Case says of duodenal diverticula: "Usually considered as without clinical significance, it is shown that they are often associated with ulcer of the duodenum or disease of the gall bladder or pancreas and when the seat of diverticulosis they may, themselves, constitute a serious menace to the patient."

While the greater number of diverticula, in both large and small intestine, apparently exist without producing any clinical evidence of their existence, trouble is caused by a sufficient number of them to make the subject an important one.

Diverticula may occur singly or in large numbers. In the duodenum, they are most often single, while in the remainder of the small intestine and in the colon, they are generally multiple. Sometimes large numbers of diverticula are present. In one specimen in the pathologic department of the New Orleans Charity Hospital, which I was permitted to examine through the courtesy of Dr. Hauser, seventeen diverticula were counted in the jejunum and ileum and ranging in size from that of a pea to that of a hen's egg. Specimens are on record where forty or more diverticula have been found.

Diverticula may be located on any part of the intestinal surface, but, that part of the intestinal wall where the mesentery is attached and the blood vessels, etc., enter and leave the gut wall, is considered, by many, a point of least resistance. It is along this border that probably the great majority of diverticula are to be found. Formerly, it was thought that this was what might be termed a point of least resistance and a natural site for diverticula. Chlumsky[3], quoted by Beer, seems to have disproven this theory. "When he injected normal small intestines that were still alive, not removed from the normal animal, the rupture occurred opposite the mesentery, whereas, as soon as he worked with intestines removed from the animal, dead some hours or more, they ruptured into the mesentery."

There are no symptoms or signs indicative of a diverticulum. The symptoms are dependent upon the portion of the bowel involved and such local changes as may have occurred. They may range from slight, indefinite abdominal symptoms to those of acute intestinal obstruction. There may be no clinical evidence of their existence or the patient may present the picture of intestinal carcinoma. In the case of duodenal diverticula, one may be very much puzzled to differentiate the condition, as the symptoms may suggest ulcer, gall bladder disease or some vague upper abdominal condition. At times, there may occur inflammatory changes (diverticulitis), adhesions and matting of coils of intestine together with a resultant obstruction. Gordinier and Sampson[4] have reported such a condition incident to diverticula in the small intestine. Diverticula occurring in the sigmoid seem particularly liable to inflammatory changes due to stasis of fecal matter in their lumen and this may lead to a train of symptoms strongly suggestive of malignancy.

The pathologic possibilities of diverticula are well summed up by Beer[5]: Diverticula which produce stenosis of the sigmoid or upper rectum.

Diverticula which lead to perforation into the peritoneum.

Diverticula that lead to abscess or localized peritonitis in the left iliac fossa.

Diverticula leading to perforation into the urinary bladder of densely adherent to the bladder.

Diverticula and mesenteritis.

Diverticula and appendicitis.

Diverticula and carcinoma.

As already mentioned, there are no symptoms or signs indicative of diverticula, therefore the diagnosis cannot be made from the history or physical examination. In the living subject, a diagnosis is dependent upon a good radiologic examination or upon the surgeon. The fact that this condition may present itself in the guise of such a variety of other conditions is good evidence of the need for a radiologic examination of the gastro-intestinal tract in all conditions (abdominal) which do not readily respond to treatment.

Examination of the duodenum should be by plate and by flouroscopy. Many times flouroscopy is necessary to properly demonstrate a duodenal diverticulum. It is my experience, that the ordinary barium meal is the most satisfactory for demonstrating colonic diverticula, the barium remaining in the diverticular pouch after the remainder of the intestine is clear.

As for treatment, it is mainly symptomatic and will depend largely upon existing conditions and any emergencies which may present themselves. In the case of colonic diverticula, however, there is one rule which should always be observed and that is to keep the feces always liquid by the use of mineral oil. By doing this we go a long way toward preventing stagnation of fecal matter in the diverticulum with the resultant inflamamtory changes, possible obstruction, etc.

REPORT OF CASES.

Case 1: Miss O. F., aged 20 years, a native of Honduras, was first seen in my office January 21, 1926. At this time she complained of epigastric pain (occurring about three hours after partaking food and relieved by food and soda.) The history was very suggestive of duodenal ulcer. Routine examination of the stool showed numerous hookworm ova present (necator Americana) and it was decided, first, to treat the parasitic condition, hoping it would prove to be the source of her trouble. She was given 4 c.c. of carbon tetrachloride and five or six subsequent stool examinations failed to show any more ova present.

Following the administration of the carbon tetrachloride, the patient's condition improved markedly and she seemed completely relieved. Six weeks later she returned, complaining of the same train of symptoms present at the time of her first visit—pain, three hours after eating and relieved by food and soda.

Feeling that the patient very likely had a duodenal ulcer, she was given a radiologic examination. By both fluoroscopic and plate examination a small diverticulum was found in the first portion of the duodenum. The filling and emptying of the diverticulum was very beautifully shown by flouroscopic study. As to the etiology, in this case, it is possible that the diverticulum may have developed at the site of a duodenal ulcer. She was placed on a modified ulcer diet and was greatly relieved of her symptoms.

Case 2: Miss E. C., white, aged 22 years, admitted to clinic at Charity Hospital Aug. 2, 1926. The complaint was stomach trouble, duration about six months. There was shortness of breath, frequent belching, "gas on stomach," poor appetite and loss in weight. Examination revealed a pulmonary tuberculosis which no doubt accounted for her complaint. During the course of the examination, radiographic examination of the colon was made to determine whether or not there might be evidence of intestinal tuberculosis. The most positive finding in the colon was a number of very beautifully demonstrated diverticula, situated in the sigmoid. These diverticula were apparently causing no trouble and were merely incidental findings.

Case 3: Mrs. F. S. M., white, aged 65 years, first seen at her home March 14, 1926. Complaint, at this time, was pain in the left lower abdominal quadrant of about three days duration, but at this time seemed to be getting better. There was a history of constipation during the attack and examination showed rather marked tenderness in the region of the sigmoid with the suspicion of a mass being present. The patient gave

a history of similar attacks occurring at intervals of six to eight to ten months and extending over a period of about ten years. Constipation was generally pronounced during an attack but during the intervals she generally felt quite well.

Here was a lady, decidedly in the cancer bearing age, with pain and a possible mass in the left lower quadrant of the abdomen and complaining of constipation. There were two favorable things in her case: she had had many similar attacks over a period of years and when first seen she had begun to improve. She was kept in bed, diet restricted to liquids and mineral oil freely administered, enemeta were also given. Within about a week, she was able to be out of bed and the pain in the abdomen had nearly disappeared. When able to leave the house, she was taken to the Hospital and radiographic examination made of the colon. This showed large numbers of small diverticula involving a large portion of the colon, but decidedly more numerous in the region of the sigmoid. What probably happened in her case was a gradual collection of fecal matter in the diverticular pouches with a resultant inflammation, and constipation.

The treatment, in such a case largely consists of one thing, mineral oil. Such patients should keep their stools constantly in a semi-liquid state by the use of petrolatum and thereby prevent stasis and other troubles resulting from it.

At times, patients are encountered, in whom such a condition is present, but in a more advanced condition and a diagnosis of malignancy might readily be made.

BIBLIOGRAPHY.

1. Nothnagel: Nothnagel's Encyclopedia of Practical Medicine, 1904.

2. Case, Jas. T.: Diverticula of the small intestine, other than Meckel's Diverticulum. J. A. M. A., 75; 1463.

3. Chlumsky (quoted by Beer, Edwin): Am. J. Med. Sc., 128; 135.

4. Gordinier, H. C. and Sampson J. A.: Diverticulitis, not Meckel's, causing intestinal obstruction. J. A. M. A., 46: 1585, 1906.

5. Beer, Edwin: Am. J. Med. Sc., 128:135.

DISCUSSION

Dr. Alton Oschsner (New Orleans): Dr. Smith has just covered the medical aspect of diverticulitis. There are several points which I should like to stress from a surgical standpoint. Although it is true that every case of diverticulitis may be prevented if the proper conservative therapy is instituted early enough, many patients are seen with advanced processes which are in reality complications of the original diverticulum. Of the complications, the most important to the surgeon is peridiverticulitis, especially with abscess formation. Localized abscess formation occurs more frequently than does a generalized infection of the peritoneum because of the preceding peridiverticulitis. These abscesses, unless recognized early and properly treated, are especially prone to rupture into an abdominal viscus, the bladder being the common viscus that is most frequently involved. Bryan found that in a series of 42 vesicocolic fistulae the rupture of a peridiverticulous abscess was the most frequent cause of fistulae. The surgical treatment of such a case consists of early recognition and prompt evacuation and drainage of the abscess. Another important complication is carcinoma. Mayo found that in 42 cases in which a resection had been performed a carcinoma coexisted in 31 per cent. Clinically, it is often impossible to distinguish between the two lessons, but this is of relatively little importance because of the high incidence of carcinoma in diverticulitis, which necessitates extirpation of the diseased portion of the bowel in all cases in which the diagnosis is not correct.

Dr. L. J. Menville (New Orleans): The radiologist is frequently called upon for help in the diagnosis of diverticulitis and both the meal and barium methods should be used. I cannot agree with Dr. Smith in accepting the meal method alone in the radiological diagnosis of this condition. The haustral segmentations of the colon will often contain remnants of the meal when the colon is nearly or completely empty, using the meal method, and these haustral markings are often mistaken for diverticula.

Dr. Smith is to be complimented in presenting such an interesting and well prepared paper.

Dr. E. D. Fenner (New Orleans): This paper is so interesting that I cannot resist the temptation of adding, as a matter of clinical experience, a synopsis of two cases of intestinal diverticula in the form of Meckel's diverticulum, that came under my observation and care.

The first case dates back a good many years. A boy of seven or eight years was brought into the hospital presenting symptoms of partial intestinal obstruction and also what looked like symptoms of severe appendicitis I operated on him and, to my great surprise, found that what I was dealing with was a Meckel's diverticulum whose tip was attached to the abdominal wall over which a loop of bowel was partly strangulated. Ligation and resection of the Meckel's diverticulum was followed by recovery of the patient.

The second is a more recent case. About four years ago a negro boy, two and a half years of age, was sent down here with a fecal fistula discharging from the umbilicus. A good deal of pus, and, every now and then, fecal matter would escape from this umbilical opening. He gave a history of infection that formed a tumor. The doctor who treated him in the country found a small abscess, made an incision and evacuated the pus. The child recovered, but was left with a fistula. I cut down around the fistulous opening through the skin, prolonging the incision at either end and had comparatively little difficulty in getting down to the intestine. At the base of a Meckel's diverticulum fecal matter was escaping. This case, I am happy to say, also recovered.

Dr. D. N. Silverman (New Orleans): Duodenal diverticula, like all other types of diverticula, are interesting. Sometimes we see cases that have undergone one or more operations and notwithstanding that the gall-bladder and appendix has been removed, these patients return to use with symptoms of upper abdominal pain. These symptoms, many of us believe, are caused by duodenal diverticula, and since the subject has been called to our attention, more than ever before, we have had occasion to see, on re-examination of roentgen-ray plates taken several years before, the presence of duodenal diverticula.

Diverticula of the colon is not always associated with constipation and the necessity of giving your patient mineral oil; quite often the opposite is the case and diarrhea is the result of the inflammatory process. Apparently those who most frequently have trouble with diverticulitis of the colon are the older individuals where there is an associated carcinomatous condition.

POST-PARTAL CARE.*

WALTER EDMOND LEVY, M. D.,†

NEW ORLEANS

Aleck Bourne makes the statement that ante-natal care is the most important single line of forward movement of thought in obstetrics in the last ten years. While, with this statement, perhaps every obstetrician is in accord, I must say that, as a rule, far too little thought and attention is given to post-partal treatment. Unless some very definite complication arises, the casual accoucheur is only too likely to let the after-care of the parturient take care of itself. While to the majority of you, I shall not bring out any very new facts, I shall attempt to outline a rational post-partal line of therapy which we have standardized on the Obstetrical Division of Touro Infirmary, and which we believe has greatly simplified matters and has given us excellent results.

Now, the first point to consider is that at the termination of the third stage of labor, viz., the birth of the placenta, the woman is, strictly speaking, in the post-partal period, and our chief concern at this movement is the prevention or control of post-partal hemorrhage. Mismanagement of the third stage of labor is one of the greatest causes of post-partal hemorrhage for the too early employment of the Crede method will cause the expression of the placenta before the uterus is ready to contract and retract. Now, if this occurs, or even if the case is handled correctly, it is our desire to control all post-partal bleeding, and to reduce it to a minimum. The technic employed at present is as follows: Immediately upon the termination of the third stage, one c. c. of pituitrin and one c. c. of gynergen (ergotomin tartrate) are given by needle. Twelve hours later, one tablet of gynergen is given. Until recently, we had been using one drahm of

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fluid extract of ergot, every four hours for six doses. Now when one stops to consider that ergot contains three alkaloids, viz., ergotoxin, histamine and tyramine, and that ergotoxin, or ergotomine is the specific alkaloid, it can readily be seen that it is highly desirable to give the specific drug.

It is very definitely stated by Dale and Dixon, that tyramine has a pressor action similar to, but weaker than adrenalin. Furthermore, Rucker has shown that adrenalin will relax a tonically contracted uterus. Bourne and Burn, experimenting with tyramine, state that it is practically without therapeutic action. As regards histamine, they state that its only effect was to increase the frequency of the contractions already present, for a short time. The effect of ergotomin can best be summed up by again quoting Bourne and Burns, who state: "An injection of 1.0 mgm. ergotomin tartrate produces a powerful contraction of the uterus that lasts for at least 16 hours; and hence is an ideal agent for use after delivery."

To recapitulate, it is highly desirable to keep the uterus in a state of tonic contraction for twenty-four hours after delivery, and we believe that this is best accomplished by the method outlined above. Occasionally, one hears much loose talk concerning the use of ergot, or its derivatives, as a cause of afterpains. To our way of thinking this is false reasoning, as afterpains are caused by the effort of the uterus to expel something, most likely a blood clot, and one can readily see that if the uterus is kept tonically contracted, such a clot cannot form and the incidence of afterpains is greatly reduced. However, if they do occur, we find that aspirin gr. V, and codein gr. ½, are almost specific. Lately, we have been trying lumalgin, and at present are highly pleased with it and intend to substitute it for codein.

DIET.

It is our custom to give all cases full diet within twenty-four hours after delivery. The exceptions to this rule are, of course, complete tears of the perineum which are immediately repaired, and the toxic cases. We can see no valid reason why a woman who has had a baby, should not be able to eat after the effects of the anesthetic have passed. In addition to this, she is given milk to drink between meals. Furthermore, it is my particular custom, as I do the low cervical cesarian section, and as a result see very little, if any digestive disturbances, such as nausea and vomiting, and dilatation of the stomach, to give these cases soft diet on the second day.

CARE OF THE BOWELS.

In none of our cases do we follow the ancient and honorable custom of giving castor oil the day following delivery, as we believe nothing in particular is to be gained from its use. We employ a daily enema of oil and glycerine, or saline, and about the fifth or sixth day, give a mild laxative in the form of cascara or milk of magnesia, if such is indicated.

CARE OF THE BLADDER.

An empty bladder is conducive to a comfortable and satisfactory puerpuerium. Not only is a full bladder a source of discomfort to the patient, but due to its size and weight, will push an uterus into retroposition and also prevent proper contraction. We have two standing orders on our division, firstly, that all patients must be catheterized every 8 hours if unable to void. Secondly, the amount voided each time must be recorded on the nurse's record. This last point is of great importance for a patient may void very frequently, but small amounts, and as a result have a full bladder.

CARE OF THE BREASTS.

In a previous communication, before this Society, on Breast Abscess, I gave in much detail the care of the breast in the puerpuerium. Suffice it to say here,

however, that the breast must be properly supported so as to obtain free drainage of the milk from the lacteals. For this purpose, we use the adhesive straps as suggested by Jellinghaus, in place of the cumbersome breast binder. Care should also be taken of the nipples so as to prevent cracks and fissures. They should at all times be covered and protected with a clean dressing. Before each nursing they should be carefully cleansed with boric acid solution, followed by sterile water. After each nursing, the nipples should be washed with 70 per cent alcohol and then sterile water, and the clean dressings reapplied. If the nipples are sore, I prefer the use of an ointment of 5 per cent resorcin in lanolin.

CARE OF THE VULVA AND PERINEUM.

After each bowel movement or emptying the bladder, the perineum and labia should be well irrigated, dried, and a fresh vulva pad applied. This particular phase in the after-care is of the utmost importance when suturing has been done.

It is our custom after delivery to allow the patients to move about freely in bed. No matter if the case be one of complete laceration, are legs tied together, as was formerly the custom, nor at any time is a case compelled to lie flat upon her back. Free moving and turning about is rather to be encouraged from two standpoints. Firstly, inactivity favors a retardation of the venous circulation, and this in turn, particularly, if associated with a severe post-partal hemorrhage, is one of the predisposing causes of thrombophlebitis. Secondly, if the patient is compelled to lie flat upon her back, the large boggy uterus, with its relaxed ligaments, easily tends to go into a position of retroversion, and if not corrected, can be a great source of future trouble. To obviate this, it is our plan to require that each and every patient, from the second day after delivery, lie for at least one hour in the morning, and one hour in the evening, flat upon her abdomen. As a result of this simple little procedure, we have cut down our incidence of post-partal retroversion enormously, and we do not find it necessary to require the patients to assume the terribly uncomfortable knee-chest position.

Patients are allowed to have the backrest after the third day, but if possible, primipara are kept in bed for a full two weeks, and multipara, for some ten to twelve days.

POST-PARTAL EXAMINATION.

Some five to six weeks after delivery, all patients are examined. Notes are made as to the condition of the vagina, the size, consistency, and position of the uterus, and the condition of the adnexa. If the uterus is retroverted, now is the time to correct it, before it becomes permanent; for this purpose, a Hodge pessary is used. No post-partal examination is complete without a visual examination of the cervix. If the cervix is eroded, everted, or lacerated, it is our custom to do a radial cauterization. This can very easily be done right in the office, or clinic without any anesthetic, local or general, care of course being taken not to touch the vaginal walls. It is my firm conviction that as a result of this cauterization, many such symptoms as leucorrhea, and the dull dragging pain in the lower abdomen (due no doubt to a para-metrial cellulitis from absorption from the chronically infected cervix), will be cured. Furthermore, as a result of the wiping out of this chronic source of irritation in the cervix, it is fair to predict that in years to come the cases so treated will show a tremendous diminution in the incidence of cervical carcinoma.

Before dismissing the subject of the post-partal examination, I should like to call to your attention one very definite symptom of which the patients very often complain, viz., backache. And to add that this is all too often blamed upon retroversion of the uterus. As a matter of fact, we find it due to, most often, one of two causes—a cellulitis in the parametrium, due to absorption

from an endocervicitis, or to a sub-luxation of the sacro-iliac syncondrosis. The treatment of the former condition has been touched upon, whereas the treatment of the latter, after confirmation, by means of the roentgen-ray, is a properly fitted sacro-iliac belt.

In closing, I should again like to emphasize the fact that post-partal care is of the utmost importance in the proper handling of an obstetric case, and is just as much of a prophylactic measure as ante-partal care, and as such, should be stressed in our teachings to medical students.

CASE REPORTS AND CLINICAL SUGGESTIONS

VOICE WITHOUT A LARYNX.*

HOMER DUPUY, M. D.,

NEW ORLEANS.

This patient which I present, a white male, aged 42 years, had carcinoma of the larynx. I did a laryngectomy in April, 1929, at Charity Hospital. An uneventful recovery followed the operation. He returned several times for observation. His return this October, 1929, proved eventful. House-officer, Dr. McNair, Department of Ophthalmology, Otolaryngology, informed me that the patient could speak in a loud voice. Let us now hear him demonstrate this to you (patient speaks to the audience in a very audible voice.)

This is surely a vocal phenomenon. Deprived of a larynx, voice seems impossible without the help of an artificial voice apparatus which has been invented, and in recent years has been perfected. Our patient has no such help. He discovered his own method of vocal production. He does it by ventriloquism. He swallows air, and by forceful diaphragmatic and chest muscle action expels air from his stomach. These air-waves become sound-waves when they are reinforced by the sounding-board apparatus of the whole head. He does not use air-currents from his trachea, as this tube is entirely cut off, by a wall of fibrous tissue.

Let us briefly review what we know relative to voice production. The vocal cords only initiate the very first step, physiologically, in phonation. Many more elements are brought into play in regions away from the larynx. The anatomical structures of the head which materially take part in voice production are, the tonsillar pillars, soft palate, tongue, teeth, accessory nasal sinuses, nasal chambers. And the reception of sound by the ear with the perception of tone variations is an other added factor in this highly complex arrangement for the production of voice, speech, and song. And again, superimposed upon this anatomic group of structures is the dominant psychologic element which also enters into voice production. We alter the tones, pitch of our voice by mental efforts. The mere mechanical dimensions of our larynx, large or small diameters, gives the low type or high type voice. But, the special peculiarity, color, individuality, of the human voice, is a question of overtones. And these are produced in the anatomic parts in the head, above the vocal cords. What makes our voice individual, something entirely different from any other voice in the wide world? It is the many modifications of these sound waves by the already enumerated structures localized in the head proper. Observation and experimentation have for several years reached the conclusion that this is the way by which the human voice is produced. Our patient furnishes a practical demonstration of this scientific fact. He reports that this is his own speaking voice of the past, before the laryngectomy. His wife, who should know, for many reasons, the voice of her mate, concurs in this report. This is "her man." The vocal results in this patient present many angles of real scientific interest to the voice teacher, physiologist, laryngologist. We

*Read before the Orleans Parish Medical Society, October 14, 1929.

esteem it a rare good fortune, that such a vocal phenomenon should have come within our ken for study and observation, and that this society is the first to hear my presentation of an extraordinary instance of "Voice Without a Larynx."

PURPURA HEMORRHAGICA IN PREGNANCY.

H. R. SHANDS, M. D.

JACKSON, MISS.

The modern conception of purpura hemorrhagica was clearly and fully presented before this Society by Cohn and Lemann in 1923. The majority of writers agree to the conclusions of Kaznelson. Keismann states: "This disease is due to a disturbance of the physiologic function of the spleen. The thrombopenia however it develops is probably due to changes in the cellular system scattered through the spleen, liver and certain lymph glands." In writing of purpura hemorrhagica Dr. Will Mayo states, "Patients are alternately better and worse, but always show evidence of the disease which eventually terminates in death." The value of splenectomy as a means of cure for true purpura hemorrhagica has been adequately proven in approximately one hundred cases.

The characteristic symptoms of purpura hemorrhagica are: 1. Petechiae and ecchymoses. 2. Bleeding from mucous membranes. 3. Prolonged bleeding time. 4. Greatly reduced platelet count. 5. Normal coagulation time. 6. Non-retractile clot.

The occurrence of purpura hemorrhagica as a complication of pregnancy is quite rare. Most of the modern text books on obstetrics do not mention it. Hirst says, "Purpura hemorrhagica is generally rapidly fatal, always interrupting the pregnancy. The foetus dies before the interruption of the pregnancy." There is no evidence that pregancy predisposes to purpura, though it is a much more serious and fatal disease when occurring during pregnancy.

Rushmore made a thorough review of the literature in 1925, and found forty-seven reported cases of purpura complicating pregnancy. Some of these cases were simple purpura. The maternal mortality in this series was sixty per cent. Dr. Rubin, in discussing Rushmore's paper, states: "This condition is now divided into two types from a prognostic point of view. One is acute purpura hemorrhagica which is practically always fatal, and the other is chronic purpura hemorrhagica which some patients enter into if they are lucky enough to escape the acute attack. For the acute attack there is nothing to do."

CASE REPORT

Report of a case of true acute purpura hemorrhagica occurring during pregnancy with recovery. Mrs. E. V., aged 25 years, was brought to the Baptist Hospital by Dr. Parkes of Union on June 17, 1929. In the past history she described several attacks of influenza, and one attack of tonsillitis fifteen years ago following which her tonsils were removed. She began to menstruate regularly and normally at 14 years. At 16 years she had small hemorrhagic spots over her body. These disappeared after two or three days without treatment. She has had hemorrhagic spots on her body, especially during menstruation, at various times since she was 16 years old. Two years ago she had more trouble than usual with larger spots on her body and for the first time bled slightly from her gums, though this condition had not been at all serious until the onset of the present illness. Her father and mother are living and well with no history of any blood dyscrasia. Two years ago one sister was treated by Dr. Eshleman in New Orleans for a chronic purpura hemorrhagica. Under medical treatment she made no improvement in six weeks. At the end of this time Dr. Eshleman had her spleen removed and she made a prompt recovery and is still well.

This patient, the mother of two children, is now six and one half months pregnant. On June 15, 1929, she had a hard rigor followed by high fever. On June 16 she began to bleed from her gums and developed marked sub-conjunctival hemorrhage. She vomited blood on June 15 and 16. On the morning of June 17 her urine appeared to be pure blood.

On arrival her temperature was 104°, blood pressure 80, pulse 120, and she presented the picture of a desperately ill woman. Her tongue and lips were swollen and hemorrhagic. She had numerous hemorrhagic spots over her entire body. Her urine was so bloody that for two or three

days her bladder had to be irrigated to remove the clots. There was also vaginal bleeding. Blood count: Hemaglobin 53, bleeding time 8 minutes, coagulation time 3 minutes, red blood cells 3,560,000, leukocytes 1,100, platelets 24,000, small lymphocytes 78, large lymphocytes 8, neutrophils 14.

Brief report of progress record for the next week: On June 17, 1929 the patient was admitted in a critical condition bleeding from nearly all mucous membranes, hemorrhagic spots over body, blood shot eyes, blood clots in bladder. Given 700 c. c. of blood, citrate method, from husband. Roentgen-ray treatment over spleen for ten minutes. Coagulen and urotropin given. Temperature 104°.

June 18: Condition unchanged except temperature 101 4-5°. 60 c. c. of blood given by transfusion. Bladder irrigated to get rid of clots. 5 c. c. of 10 per cent calcium chloride given intravenously. Severe pain in abdomen. Vomiting large quantities of dark fluid. Mouth wash was used. Morphin gr. ⅛ P. R. N. Amount of urine diminished.

June 19: Gums stopped bleeding. Urine very bloody, no clots. Less pain. Temperature 100°. Uncomfortable night. Roentgen-ray treatment over spleen for ten minutes. 5 c. c. calcium chloride given intravenously.

June 20: Temperature 99°. Lips and tongue improving. No bleeding except bloody urine. Better night. 5 c. c. calcium chloride intravenously. B. P. 90. 700 c. c. of blood given by transfusion. Pain in back and abdomen.

June 21: Temperature 100½°. Has had marked pain and cramping in abdomen. Roentgen-ray treatment for ten minutes over spleen. Quinin bisulphate ten grains by rectum every eight hours. 5 c. c. calcium intravenously. Hemaglobin 62, red blood cells 3,830,000, leukocytes 3,900, lymphocytes 61, platelets 65,000.

June 22: Urine normal in color. Temperature normal. Hemorrhagic spots clearing up. 600 c. c. of blood by transfusion. General condition better. Nourishing nicely. Roentgen-ray treatment ten minutes over spleen.

June 24: Condition improved. 700 c. c. of blood by transfusion. Clotting time and bleeding time 3 minutes. B. P. 95 over 35. Platelets 85,000.

June 25: Still improving. Sleeping well. B. P. 100 over 40. No nausea. No pain. Urine normal. Hemaglobin 64, coagulation time 2½ minutes, red blood cells 3,810,000, white cells 4,400, bleeding time 3 minutes, platelets 90,000.

By June 27, ten days after admission, with the exception of a secondary anemia and low blood pressure and platelet count, she was approximately normal. During the ten days she was given 4,000 c. c. of blood, citrate method, no reactions, four roentgen-ray treatments, and five doses of calcium chloride intravenously. Although she had been given one gallon of blood by transfusion the platelets had only risen from 24,000 to 95,000 and blood pressure from 80 to 100. On this date at 9:00 a.m. she was operated upon under ether anesthesia. A long midline skin incision was made and the fascia first opened below the umbilicus and an abdominal hysterotomy done. A six and one half months living foetus was removed, after giving pituitrin and gynergen. There was no abnormal bleeding. The uterus was sutured and the incision in the fascia extended upward to near the ensiform. The spleen was then quickly and easily removed.

The time of the complete operation was thirty-five minutes. During the operation a blood transfusion and intravenous saline was given. There was no shock and very little hemorrhage. The patient was returned to her room in good condition and made an entirely uneventful and complete recovery, being discharged from the hospital eleven days later. By 5 p. m. on the day of operation the platelets had risen to 165,000. On June 28 the platelets were 220,000. On July 4 the platelets were 290,000 with hemaglobin 81 and red blood cells 4,500,000.

She was examined again on September 30 at which time she was quite well with a normal blood picture. The wound was firmly healed with no evidence of hernia. Her strength was so good that she voluntarily walked to the third floor of the hospital for the blood count instead of waiting for the elevator. She was seen again on November 30 at which time she was in excellent health. The foetus died three hours after delivery.

We were lead to adopt the above outlined preparatory treatment for the acute purpura by experience in two former cases. J. B. admitted to the Baptist Hospital February 21, 1929, with acute purpura with a temperature 104½°. Two weeks before this he had been seen in the office with a mild purpura. He was given two blood transfusions and this condition cleared up promptly. We advised splenectomy at this time but the patient's family were not inclined to acept it. The acute phase developed two weeks later. Blood transfusions alone did not control the acute phase and he died three days later, temperature remaining around 104. H. C., aged 3 years, admitted to Baptist Hospital December 12, 1927, in a desperately critical condition, with profuse hemaglobinuria and very severe anemia. Red blood cells 1,500,000, hemaglobin 30, white cells over 100,000. He presented a blood picture of a terrible blood destruction, with marked anisocytosis, poikilocytosis and many normoblasts, temperature 101°,

pulse 140. A diagnosis of acute leukemia was made and repeated blood transfusions and roentgen-ray treatments over spleen given—spectacular recovery—discharged five days later. Now two years later the boy is still in excellent health with a normal blood picture.

In 1919 Stephan of Prague reported two cases of purpura hemorrhagica fulminans as cured by roentgen-ray treatment over the spleen. A few observers have reported similar happy results in single cases. Pancoast and his associates in 1925 were unable to duplicate the results of Stephan in any of six cases reported, none of their cases being cured, though five or six were temporarily improved by the roentgen-ray treatment. Some of their cases were also given blood transfusions. As a means of permanent cure they place no value on roentgen-ray treatment alone.

Larrabee of Boston has urged the advisability of large transfusions frequently repeated, in attempting the control of the acute stage of purpura. Whipple reports most satisfactory results in the treatment of chronic thrombocytopenia by splenectomy. He reviews seventy-three cases with six operative deaths and good late results in eight per cent of the followed cases. He states that the acute type is not as a rule saved by splenectomy. He found eight cases of purpura fulminans treated by splenectomy during this stage with seven operative deaths. These fulminating cases should certainly be given some treatment calculated to tide them over the acute stage so that a splenectomy can be safely done a little later.

In the September, 1929, number of the Surgery, Gynecology and Obstetrics, Deaver recommends the combination of blood transfusions, roentgen-ray over the spleen and calcium chloride intravenously in severely jaundiced cases in preparation for operation. This was exactly the combination of treatment given by us the preceding June and was successful in promptly controling a desperately serious case of purpura hemorrhagica so that a splenectomy could be safely done. While this is only one case the outcome was so gratifying that this method of treatment is recommended to you for your consideration and trial in a condition which has formerly been considered practically hopeless.

I find a record of only one other case of purpura hemorrhagica in pregnancy which was treated by splenectomy. This was an acute case and died.

Some criticism may be offered on the performance of the caesarean section at this operation. The reasons for so doing were briefly as follows: 1. The foetus is general'y lost in purpura hemorrhagica anyhow. 2. Roentgen-ray treatment over the abdomen would probably have unfavorably affected the foetus if it had happened to live. 3. Miscarriage had been seriously threatened in the ten days of preparation, and a six months miscarriage following a serious abdominal operation is a grave and often fatal complication. 4. The cause of death in purpura hemorrhagica in pregnancy is most often uterine bleeding during or after delivery. Mechanically the splenectomy could be much more easily and quickly done after the uterus which almost filled the abdomen had been emptied. My judgment indicated to me that I could more safely handle the case in this way.

BIBLIOGRAPHY.

Pancoast, H. K., Pendergrass, E. P. and Fitz-Hugh, T. Jr.: The present status of the roentgen treatment of purpura hemorrhagica by irridation of the spleen. American Journal of Roentgenology. 13: 558-567, 1925.

Stephan, Richard, Reticulo-endothelialer Zellapparat und Blutberinnug. (Reticulo-Endothelial Cell Apparatus and Coagulation of the Blood). Munchen med. Wchnschr., 62: 309-312, 1920.

Mayo, William J.: Splenomegalias. Collected papers of the Mayo Clinic, page 775, 1923.

Hirst: Hirst Manual of Obstetrics.

Rushmore, Stephen: Purpura Complicating Pregnancy. JoJur. Obstet. and Gynec. No. 4. October, 1925. Discussion by I. C. Reuben.

Deaver: Gall stone disease. Causes of morbidity and mortality of operation for. Surg., Gynec. and Obstet., Sept., 1929.

Keismann: Med. Klin. Jan. 1921.

Cohn and Lemann: Splenectomy as a treatment of purpura hemorrhagica. rans. South. Surg. Assoc., 1923.

Larrabee: Boston Medical and Surgical Journal.

Whipple: Splenectomy as a therapeutic measure in thrombocytopenia purpura hemorrohagica. Surg. Gynec. nd Obst. March, 1926.

REVIEWS

SURGICAL DISEASES OF THE PANCREAS*

I. M. GAGE, M. D.,†

NEW ORLEANS.

EMBRYOLOGY.

The pancreas arises, a little later than the liver, as two or three separate outgrowths, one from the dorsal surface of the duodenum usually a little above the liver outgrowth, and one or two from the lower part of the common bile duct. Of the latter outgrowths, that upon the left side may be wanting, and if formed, early disappears, whereas that on the right side continues its development to form what has been termed the ventral pancreas. Both this and the dorsal pancreas continue to elongate, the latter lying to the left of the portal vein, while the former, at first situated to the right of the vein, later grows across its ventral surface so as to come in contact with the dorsal gland with which it fuses so intimately that no separation line can be distinguished. The body and tail of the adult pancreas represent the original dorsal outgrowths, whereas the right ventral pancreas becomes the head.

Both the dorsal and ventral outgrowths early become lobated, and these lobes becomes secondarily; by this lobation repeating itself several times the compound tubular structure of the adult gland is acquired, the very numerous terminal lobes becoming the secreting acini, and the remaining portions becoming the ducts. Of the principal ducts, there are at first two; that of the dorsal pancreas, the duct of Santorini, opens into the duodenum on its dorsal surface, whereas that of the ventral outgrowth, the duct of Wirsung, opens into the choledochus. When the fusion of the two portoins of the gland occur, an anasomosis of branches of the two ducts develops; the proximal portion of the duct of Santorini may degenerate, so that the secretion of the entire gland empties into the common bile duct through the duct of Wirsung.

In the connective tissue which separates the lobules of the gland, groups of cells occur, which have no connection with the ducts of the guand, and from what are termed the islands of Langerhans. They arise by a differentiation of the cells which form the original pancreatic outgrowth, and have been distinguished in the dorsal pancreas of the guinea pig while it is still a solid outgrowth. They gradually separate from the remaining cells of the outgrowth and come to lie in the mesenchyme of the gland in groups, into which, finally, the blood vessels penetrate(1).

FUNCTION OF THE GLAND.

The pancreas, by its system of ventral connections with the gastric intestinal tract, passes its secretion into the alimentary canal, in order that the digestive process may proceed normally. The secretion of the pancreas is composed of digestive ferments which are actuated when entering the intestinal canal and aid in the digestion of fats, proteins, and carbohydrates.

The greatest function of the pancreas comes from its internal secretion, insulin, which governs carbohydrate metabolism. Therefore, in disease of the pancreas we have interference with digestive processes and the metabolism of carbohydrates, and these conditions are manifested through the organism as signs and symptoms of a diseased state of the pancreas.

ANATOMY OF PANCREAS.

The pancreas is a compound racemose gland, analagous in its structure to the salivary glands, though softer and less compactly arranged. It is long, irregular, and prismatic in shape, and of a reddish

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white color. Its right extremity, being broad, is called the head, and is connected to the main portion of the organ, the body, by a slight constriction, the neck, whereas its left extremity gradually tapers to form the tail. It is situated transversally across the posterior wall of the abdomen at the back of the epigastric and left hypochondriac region. Its length varies from five to six inches, its breadth is an inch and a half, and its thickness from half an inch to an inch, being greater at its right extremity and along its upper border.

The head is lodged within the curve of the duodenum. The upper border is in contact with the first portion of the duodenum, its lower overlaps the third part, and its right and left borders overlap in front and insinuate themselves behind the second and fourth parts of the duodenum, respectively. In the groove between the duodenum and the right lateral lower borders in front are the anastomosing superior and and inferior pancreatic-duodenal arteries; the common bile duct descends behind, along the right border, to its termination in the second part of the duodenum. The anterior surface is in relation to the transverse colon, gastroduodenal artery, superior mesentery artery and vein. The posterior surface is in relation with the inferior vena cava, the renal veins, the right crus of the diaphragm, and the aorta.

Blood Supply: Superior pancreatic duodenal branch of the gastroduodenal; the inferior pancreatic-duodenal branch of the superior mesenteric; the inferior pancreatic branch of the superior mesenteric; pancreatic branches of the hepatic and pancreatic branches of the splenic arteries.

Lymphatics: The lymphatics arise in a network within the lobules. Numerous collecting trunks pass to the surface of the pancreas. The splenic nodes receive most of the turnks. Others are received along the aorta, nodes at the origin of the superior mesenteric artery, and nodes along the pancreatic-duodenal vessels. There is anastomosis of lymphatics with those of the gallbladder.

Veins: The veins are the anterior pancreatic-duodenal branch of the superior mesenteric, the posterior pancreatic-dudenal branch, and the pancreatic branches of the portal, and the pancreatic branches of the splenic.

Nerves: The nerve supply is derived from the coeliac, superior mesenteric, and splenic plexuses.

DISEASES OF THE PANCREAS.

I. Injuries to pancreas.

II. Inflammatory conditions of pancreas.
   - A. Acute pancreatitis.
     1. Acute hemorrhagic.
     2. Gangrenous.
     3. Suppurative.
   - B. Sub-acute.
   - C. Chronic pancreatitis.

III. Pancreatic cysts.
   - A. Retention cysts.
   - B. Proliferous cysts.
     1. Cyst adenoma.
     2. Cystic carcinoma.
   - C. Congenital cystic disease.
   - D. Dermoid cysts.
   - E. Hydatid cysts.
   - F. Hemorrhagic cysts.
   - G. Pseudo cysts.

IV. Neoplasms of pancreas.
   - A. Adenoma of pancreas, rare.
   - B. Carcinoma of pancreas (most common new growth) (2 per cent of all cancers).
   - C. Sarcoma of pancreas, rare.

V. Pancreatic calculus.

INJURIES.

The pancreas is rarely injured. This is due in part to the deep position that it occupies and in part to the ribs and overlying viscera.

The number of cases in which the pancreas has been grossly injured is probably small; slight injuries usually escape notice. The first case found in the literature was recorded by Travers in 1827. Many associated lesions are observed, *i. e.*, rupture of duodenum jejunum, tranverse mesocolon, etc. The blow which usually causes the injury is generally applied to the epigastrium, and the direction of the force is before backward. The injury may be a crushing one as between two cars or some blunt instrument of force applied directly over the epigastrium.

The signs and symptoms are those of shock which gradually deepens, signs of hemorrhage, and in later stages signs of peritonitis.

Gunshot wounds of the pancreas during the late war were almost invariably fatal due in part to lesions of associated viscera. Moynihan has one case to recover.

Treatment: Following a severe blow or jury to the upper abdomen the operation should never be completed without an examination of the pancreas. The operative principles which must be observed, as laid down by Moynihan, are:

A. Bleeding must be carefully arrested by ligature en masse or by suture, preferably the latter.

B. If the tail of the gland is very badly crushed, a resection must be performed.

C. If the duct of the gland is exposed, care must be taken to see that the sutures do not penetrate the duct.

D. Free drainage through the anterior wound; through a special posterior wound, or drainage through both must be provided.

E. The patient should also have supportive treatment. Here transfusion and glucose preoperatively are indicated, and postoperatively glucose plus insulin.

INFLAMMATORY CONDITIONS OF PANCREAS.

Infection may reach the pancreas (1) by way of the lymphatics, (2) through the ducts, (3) through the blood stream, or (4) by contiguity.

In 1908 Maugeret[4] first demonstrated the connection of the efferent pancreatic lymph vessels with the efferent lymph vessels of the gall-bladder around the head of the pancreas, and believed that infection could easily gain access to the pancreas by this route.

Braithwaite,[5] of London, has also shown that there is a connection of the lymphatics of the ileo-cecal region with those around the head of the pancreas and the ducts of the gall-bladder, and Wilkie[6] has shown that there is 85 per cent involvement of the lymphatics along the gall-bladder ducts in acute and chronic infections of the gall-bladder.

The term "pancreatic lymphangitis" was applied to this condition by Arnsferger in 1911. Deaver and Pfeiffer,[7] a year later, reported a detailed study upon the subject and considered it a process which precedes true interstitial pancreatitis.

In 1911 Franke[8] demonstrated the anastomosis of the lymphatics of the gall-bladder with those of the head of the pancreas by means of Gerota's injection method, and found a node in the neck of the gall-bladder which communicated with the lymphatic vessels of the pancreas. This observation is important, since this node is enlarged frequently in cases of chronic pancreatitis. Bartels[9] also pointed out that lymphatics of the head of the pancreas and duodenum are very closely related and possess no intervening lymph nodes, and Franke demonstrated the same condition in the efferent lymphatics of the gall-bladder.

If a block should occur further down in the lymphatic vessel, a reversal of the lymph flow may take place and flow into the efferent channels of the pancreas. In this way infection may take place.

Deaver maintains that chronic pancreatitis is the result of an infection of the interlobular connective tissue of the pancreas and, therefore, originates as a lymphangitis. The infection in some of the acute cases is carried by the lymphatics, as the disease is not always associated with the lodgement of a gallstone in the ampulla of Vater, causing retrojection of bile into the pancreatic duct, and furthermore, in a fair percentage of the cases gallstones are not present.

The fact that chronic pancreatitis is most frequently confined to the head, and that the common duct passes through the head in 80 per cent of the cases, whereas the tail is relatively immune, lends considerable weight to the lymphatic route.

Deaver and Pfeiffer, in examining 51 cases of chronic pancreatitis, discovered the head alone to be affected in 42; in 9 cases only was the tail involved, and in 27 cases there was evidence of involvement of the lymph nodes at the head of the pancreas.

The existence of a localized hepatitis in the vicinity of the gall-bladder, in addition to pancreatitis, gave rise to the opinion that there was a close association between the lymphatics of the liver, gall-bladder, and pancreas. Graham and Peterman[10] (1922) injected organisms into the portal vein in order to produce infection of lymphatics of liver. Examination of the biliary tract at autopsy revealed injection of the gall-bladder, common duct, and pancreas, and a definite lymphangitis was shown to exist in the pancreas by histological study.

In 1927 Kaufman[11] repeated these experiments and found the same lesions as Graham and Peterman, but in addition he noted changes in other organs—*i. e.*, the spleen, kidney, heart, and lungs. The recovery of the organism from the heart's blood in pure culture led him to believe that he was dealing with a bacteremia.

Kaufman produced cholecystitis in cats by injecting bacteria into the gall-bladder of the 26 animals so infected; only 2 showed bands of small round cells invading the pancreas. As Ordway[12] described the same appearance in normal cats, Kaufman concluded that infection of the pancreas by the way of the lymphatics is unlikely. However, clinical experience shows that in the adult with one definite pathological lesion there are usually other pathological states associated. In chronic appendicitis there is frequently an associated chronic cholecystitis. Chronic cholecystitis seems to be the etiological factor in a large number of cases of chronic pancreatitis. It is probable that the lymphatic arrangement in the cat is not comparable to that of an adult human being.

ACUTE INFECTION OF PANCREAS.

(1) Acute hemorrhagic pancreatitis.
(2) Gangrenous.
(3) Suppurative.

In acute pancreatitis the condition is entirely different from the chronic form. In the latter type the pathological picture is of a slow progressive type, and produces round cell infiltration and fibrosis. There is no activation of the pancreatic ferments, therefore no destruction of the gland tissue.

In the acute type of pancreatitis there is activation of the pancreatic ferments, setting free in the surrounding tissues trypsinogen derived from gland cells. The transformation of trypsinogen into trypsin occurs in one or two ways: (1) Enterokinase, as may happen rarely in coincident injury of the pancreas and intestines, (2) an activating substance produced when the gland itself undergoes autolysis. It is known from the post-mortem digestion of the pancreas that the substances necessary to bring about autolysis of the pancreas is liberated by death of the pancreatic cells,

and that this substance activates the trypsinogen. This is illustrated by the introduction of a normal pancreas into the abdominal cavity of a normal dog which will produce death within twenty-four hours, autopsy showing the picture of acute hemorrhagic pancreatitis.

The theory as to the cause of acute hemorrhagic pancreatitis has centered around the introduction of bile into the pancreatic duct. Claude Bernard, in 1856, when he injected bile and sweet oil into the pancreatic duct, produced acute necrosis of the gland.

In 1899 Lanereaux[13] suggested that gall stones lodged in the ampulla of Vater might cause pancreatitis by obstructing the pancreatic duct.

Opie,[14] in 1901, reported a case in which necropsy revealed a gall stone lodged in the ampulla of Vater. The stone obstructed the outflow of bile into the duodenum, but did not prevent its regurgitation into the pancreatic duct. This observation was followed by many comprehensive studies to show that the retrojection of bile into one pancreatic duct would or would not produce acute hemorrhagic pancreatitis.

Flexner,[15] Carnot, Opie, and others showed that the retrojection of bile, especially the bile salts (Flexner) would produce acute hemorrhagic pancreatitis.

Polya,[16] in 1912, indicated that acute pancreatitis was not as likely to occur following injection of duodenal contents, infected bile, or bacteria as when a very active solution of trypsin was used. In 1913 Nordman[17] showed that it was necessary to tie both ducts to produce stasis of pancreatic secretion within the ducts of the gland before pancreatitis would develop. Archibald and Gibbons[18] (1921) found when normal bile was injected into the pancreatic duct that an aseptic necrosis resulted, whereas the injection of infected bile produced more congestion, edema, hemorrhage, and fat necrosis. The observations of Flexner, Polya, and Nordman suggest that activation of the pancreatic juice is of a chemical nature, and that infection may play a role in the production of the chemical agent. Moynihan believes that the immediate cause of the hemorrhage necrosis is by activation of the pancreatic secretion by some agent within the gland substance.

Egdah,[19] in a series of 105 post mortem examinations in acute pancreatitis, found 32 cases associated with gastro-duodenitis and 17 with a history of chronic alcoholism.

Archibald thinks that the retrojection of bile into the pancreatic duct is the cause of acute pancreatitis, and thinks the mechanism by which this is brought about is by a spasm of the sphincter of Oddi.

In 1921 Mann and Giordano[29] investigated from anatomical and experimental aspects the role played by the bile in producing acute pancreatitis. The anatomic relations of the common duct to the duct of Wirsung were carefully reviewed in 200 consecutive autopsies. In 3.5 per cent of the cases it was found that it would be anatomically possible for obstruction at the ampulla to convert the two ducts into a continuous channel and permit bile to pass into the pancreatic duct. They concluded that the percentage of cases in which the two ducts could be converted into one by the contracture of sphincter of Oddi would be very small. In 1921 Judd studied the question anatomically, in order to determine the percentage of instances in which the two ducts could be converted into one, either by a stone obstructing the ampulla of Vater or spasm of the sphincter of Oddi. He found that this was possible in 4.5 per cent of the bodies examined. Mann showed that the bile would have to be under pressure of 800 mm. of water and that in all cases it passed into the duct at less than 500 mm. Bile was injected into the duct at pressures simulating that which could possibly occur in the common duct and no pancreatitis developed except in one case in which there was fat necrosis. When bile was injected with a syringe into the duct, the pressure

being much greater than normal, typical hemorrhagic pancreatitis and death followed in many instances.

Acute infection of the pancreas by way of the blood stream is rare, although chronic pancreatitis, which may result from toxic or constitutional disorders carried by the way of the blood, is more frequent. Pancreatitis occasionally develops in the course of acute infectious diseases; and Deaver has noticed an incidence of 6.5 per cent occurring in epidemic diseases. However, the condition is not serious, as they found one death attributal to that etiological factor.

At times the pancreas becomes infected by contiguity of tissue, especially rupture of duodenal and gastric ulcers. Ochsner[21] reported a case of abscess of the pancreas, following a rupture of a gastric ulcer on the posterior wall of the stomach. Abscess of any of the adjacent viscera may extend to and involve the pancreas.

SIGNS AND SYMPTOMS OF INFLAMMATORY CONDITIONS OF THE PANCREAS.

Symptoms: In chronic pancreatitis the symptoms are indefinite. It is rarely met with before the age of 40, although it may occur earlier. The symptoms are usually referred to related viscera, that is, those pathological states that occur in the gall-bladder and liver, and occasionally the appendix. The symptoms are usually referable to symptoms of chronic gall-bladder disease, which probably is the most frequent forerunner of chronic pancreatitis. The passage of bulky, soft, fetid stools, containing undigested fat and protein, also aids in arriving at a diagnosis of chronic pancreatitis. One of the most difficult diagnostic problems is that of differentiating chronic pancreatitis of a severe degree from carcinoma of the pancreas. This is true clinically as well as at operation.

Treatment: The treatment of chronic pancreatitis is its prevention. By early recognition of processes within the appendix, gall-bladder and liver, and by the removal of diseased gall-bladders and appendices, we are able to prevent the constant lymphatic absorption of toxins from these foci which invariably lead to chronic inflammatory changes in the pancreas.

Where the chronic pancreatitis has existed for some time, it becomes difficult to differentiate the condition from carcinoma if jaundice is present, which symptom sometimes accompanies chronic pancreatitis. If this condition exists, then cholecyst - duodenostomy or cholecyst - gastrostomy is the procedure of choice.

ACUTE PANCREATITIS.

This is one of the most severe calamities that occurs in connection with the abdominal viscera. Of all the pathological processes of an acute nature that occur to a human being, this is the most agonizing, and one that is attended by a high mortality.

This disease was divided into three types by Fitz of Boston, in 1889, (Med. Record, 35:197), who was one of the first to describe the condition of acute pancreatitis. The types are (1) hemorrhagic, (2) gangrenous, and (3) suppurative. The types vary only in degree, being the sequence of each pathological state.

The etiology has been discussed above. However, there are several theories. (1) regurgitation of bile into the pancreatic duct by (a) gall stone obstructing the papilla of Vater; (b) spasm of sphincter of Oddi; (2) acute lymphangitis; (3) infection through the blood stream; (4) vascular thrombosis of the pancreatic vessels.

Symptoms: The onset is sudden. There is the most severe agonizing pain, (more so than in any other abdominal catastrophe), in the epigastrum. The pain is probably the outstanding symptom, and is confined to the epigastrum at first; later the pain may be felt in the back, and even in both loins. The pain is so intense that the patient immediately collapses. The face is pallid, and the face and limbs are cold. There is never any doubt about the pres-

ence of shock in acute pancreatitis. Vomiting is almost always present, probably due to sudden infiltration around the head of the pancreas and duodenum, which Hunt[22] believes produces marked irritation of the duodenum and possible obstruction. Hunt also believes that the sudden liberation of insulin, as well as split proteins could account for the shock, the liberation of insulin producing a sudden hypoglycemia. The face may be livid and patches of a slate blue color may be distributed irregularly over the surface of the abdomen and legs. Halstead first called attention to this condition. The abdomen is rigid, being most marked above the umbilicus; even in the early hours there may be a definite fullness noted above the umbilicus. There is one laboratory method that may be of considerable aid in the diagnosis of acute pancreatitis, and that is the estimation of the diastase in the blood. The value of this test has been emphasized by Guleke,[23] Unger,[24] Hess[52] and DeTakats.[26] DeTakats advises the use of the method of Wohlgemuth because of its simplicity. In 97 cases of acute necrosis of the pancreas collected by Schmieden and pancreas collected by Schmieden and Sebering, 78 cases (80 per cent), showed abnormally high figures for diatase in the blood. DeTakats and Nathansan found high figures for diastase in the blood in their experimental work on the correlations of the internal and external pancreatic secretions.

The condition must be differentiated from ruptured gastric ulcer, duodenal ulcer, acute intestinal obstruction, and rupture of tubal pregnancy.

Treatment: Immediate operation and drainage of the pancreas is indicated. However, as suggested by Hunt, the patient should be detoxicated by giving sodium chlorid and glucose intravenously before operation, because there is evidence of high intestinal obstruction which requires treatment, as well as the sudden flooding of the patient with insulin resulting in a hypoglycemia. The same medication should follow operation, and insulin should probably be given to rest the pancreas. This treatment should be controlled by blood sugar estimations.

The surgical approach to the pancreas is through the gastrohepatic omentum, gastrocolic omentum, and transverse mesocolon, the posterior approach through the back being seldom used. The pancreas should be exposed, and the gland capsule should be opened and thorough drainage established by means of large gauze drains surrounded by rubber dam tissue. The omentum should be used to wall off the rest of the abdominal cavity. In this connection Royal Watkins[27] gives some very interesting statistics in regard to the routes used in the approach and drainage of acute pancreatitis. There were 8 cases drained through the gastrohepatic omentum, with 1 recovery and 6 death;s 3 drained through foramen of Winslow, with 1 recovery and 2 deaths; 1 drained through the common duct, with recovery.

The drainage through the gastrocolic omentum allows the suturing of the omentum to the peritoneum around the drainage tubes and prevents dissemination of the toxic products into the peritoneal cavity and their absorption. Jones, as quoted by Moynihan, reported 56 cases from the Massachusetts General Hospital, with a mortality of 60.7 per cent. The mortality of acute pancreatic necrosis without abscess formation was 72.3 per cent. Moynihan reported 21 cases from the Leeds General Infirmary between the years 1915-1924, with a mortality of 38 per cent. There were 8 males and 13 females.

Subacute pancreatitis is considered by Moynihan as occupying a position between the acute and gangrenous types. The most frequent condition found at operation is an abscess. These cases should be treated in the same manner as acute pancreatitis as far as operative procedures are concerned.

CYSTS OF PANCREAS.

Cysts of the pancreas are divided into two main groups, true cysts and pseudocysts;

the former originating in the pancreatic tissue proper, and the latter as a rule, arising in, and confined to, the cavity of the lesser sac. The pseudocysts usually follow trauma with spilling of pancreatic secretion into the lesser sac, where it is confined. The contents may be hemorrhagic or clear, depending on the amount of hemorrhage present in the cystic cavity. The hemorrhagic cysts usually follow subacute or acute hemorrhagic pancreatitis of mild degree.

True cysts usually result from one of the following conditions, which may then block the excretory duct of the pancreas, (1) stones impacted in the duct, (2) cicatricial-stenosis, (3) pressure on the duct from without, (4) dislocation of part of the gland.

The proliferation cyst may be benign or malignant. At times it is almost impossible to separate the benign from the malignant forms, pathologically.

Congenital cysts usually are observed only on rare occasions, and are similar to the congental cystic diseases of the kidneys and liver.

Hydatid cysts of the pancreas, as hydatid cysts of the spleen, are very rare.

Dermoid cysts may occur in the pancreas. Warren and Dermis[29] reported a case in 1923, and Judd[30] reported a case occurring in the tail of the pancreas in 1921.

Cysts occur more frequently in males than females.

Symptoms: The symptoms of pancreatic cyst are usually those of pancreatic deficiency and the symptoms arising from pressure on surrounding organs. The patient may be ill and complain of indefinite symptoms, indigestion, nausea, vomiting, loss of weight, dysfunction of the normal motility of the gastro-intestinal tract, constipation alternating with diarrhea, and at times stools may show blood, associated in some cases with jaundice. The outstanding symptom is a tumor in the epigastrium that is fluctuating in character and is not freely movable. There is pain of a dull character, which is confined to the epigastrium, but may radiate to the back; at times the pain is very acute. The patient shows exacerbation of symptoms which may come every two or three months. At this time all symptoms are increased in severity.

Roentgenray study of the gastrointestinal tract is a very important aid in diagnosis, as the pancreatic cyst has a definite effect on the curve of the duodenum and should be used when possible.

Pancreatic cyst must be differentiated from ovarian cyst, hydrops of gall-bladder, cysts of suprarenal capsule, omental cyst, cyst of mesentery, hydronephrosis, cyst of spleen (rare), retroperitoneal cyst, and aneurysm.

Treatment: Probably the best treatment is incision with suturing of the cystic sac to the abdominal wall. This allows free drainage. It is seldom possible to extirpate the cyst because of its attachments to important organs and blood vessels; however, in some instances it is practicable to completely extirpate the entire cyst. This should always be done whenever possible.

MALIGNANT TUMORS OF THE PANCREAS.

Cancer of the pancreas is found in about 2 per cent of autopsies on malignant tumors. It is the most frequent tumor of the pancreas. It may be secondary to carcinoma of the stomach, or from the ampulla of Vater; and, when both the duodenum and pancreas are involved, it may arise from aberrant pancreatic tissue in the duodenum.

The location of the tumor is, in the great majority of cases, confined to the head of the pancreas; however, it may diffuse through the entire organ. Ewing[31] collected 386 cases from the literature and classified their distribution as follows: 158 were diffuse, 156 limited to the head, 28 in the body, and 12 in the tail.

Although early tumors may be limited to the pancreas, they soon invade the surrounding structures. The early mestastases are to the regional lymph nodes and the liver. Generalized metastasis is not common, probably because the disease is rapidly fatal. Carcinoma of the pancreas is more common in men than women, the ratio being 6 to 1. The disease rarely occurs before 40; however, it has been found in children.

Symptoms: The first symptoms are usually vague abdominal pain and indigestion. Occasionally the onset is sudden with pain simulating gall stone colic. Judd and Parker [32] studied 34 cases of carcinoma of pancreas, and, contrary to general teaching, colic with jaundice occurred in 35.29 per cent of these cases, pain with jaundice in 23.52 per cent, and colic, pain, and jaundice occurred in 61.75 per cent, and jaundice without pain in only 38.22 per cent of cases. Intermittent jaundice ococcurred in 5 of the 12 cases with colic alone. Gall stones were found in one case. Jaundice is an early symptom, due to compression of the common duct, which passes through the head of the pancreas (in 80 per cent of individuals, Mayo, C. H.). The jaundice is progressive and lasting; cachexia and emaciation of extreme degree occur in the later states. The gall-bladder becomes enlarged and hydropic (Law of Courvoisier, 1890). Elkin[33], of Atlanta, reported 25 cases of carcinoma of the pancreas, and showed that in his series 22 (88 per cent) showed distinct enlargement of the gall-bladder.

Diagnosis: Sudden onset of jaundice, which increases in severity, clay colored stools, large, fluffy, fatty stools, undigested fat and protein found in examination of stools, loss of appetite, and emaciation occurring in a patient past 30 years is very suggestive of carcinoma of the pancreas.

Roentgen-ray at times shows increased diameter of duodenal curve.

Treatment: The treatment is palliative, mainly by short-circuiting the bile into the gastrointestinal tract by cholecystogastrostomy or cholecystduodenostomy. Where the growth is pedunculated or situated in the tail of the pancreas without metastasis, partial pancreatectomy may be accomplished with fair hope of cure.

PANCREATIC CALCULUS.

The occurrence of stone in the pancreas was first recorded by Graff in 1667. In 1883 Johnson collected 35 recorded cases. The fullest account was given by Giudiceandrea, who recorded 48 cases from the literature with a report of two personal cases, in 1896.

The stones are generally white, greyish white, or yellowish white in color, rounded, ovoid, or elongated like a date stone; facets are rarely seen on them. They are found in all parts of the ducts of the pancreas, though much more frequently in the head; in the extremity of the tail of the gland they are rarely seen. The calculi may branch like coral, the trunk of the stone lying in the main duct and its offshoots in the secondary ducts. At times the duct of Wirsung is found packed ful of stones, with sand or fine rounded pellets. As many as three hundred stones have been found in a single case. The stones are chiefly composed of phosphorus and carbon salts. Some stone may be formed of oxalate of lime stone.

Schmieden and Sebening[34] recorded a series of twenty cases of pancreatic stones, eleven of which were removed at operation. All of these patients recovered. Of the twenty cases of pancreatic stone, only two were demonstrated by the roentgen ray

Diagnosis: The diagnosis of pancreatic calculi has been rarely made preoperatively and unless constantly kept in mind when exploring the pancreas, especially when the gall-bladder shows no pathology

and the patient has symptoms of biliary stones, they may even be overlooked then.

The symptoms usually consist of presistent attacks of epigastric pain, uneasiness of the type of hepatic, colic, though less severe and unattended until very late in the history by jaundice. The pain which is in the epigastric region may at times radiate through.

The stools are sometimes frothy, characteristic of the stools seen in cases of chronic pancreas may accompany pancreatic calculi of long standing. There may be sugar in the urine accompanied by a hyperglycemia, and that diastase of the blood may be increased. Roentgenogram is of little value, as only a very small per cent will cast a shadow.

Treatment: The treatment is entirely surgical. The pancreas is exposed through an upper abdominal incision, with subsequent opening of either the transverse mesocolon or the gastro-colic omentum. The pancreas is thoroughly palpated from the head to the tail for presence of calculi. Where the stone is located near the ampullo of Vater, the transduodenal approach with splitting of the papilla will be found very useful, as the stone can then be lifted out from the duct with a scoop. When the stone is located farther back along the duct of Wirsung in the body of the pancreas, the pancreas should be incised, the duct opened, the calculus removed, the gland sutured with catgut, and drainage established. The drainage tubes should pass through the gastro-colic omentum in order that the gastro-colic omentum can be sutured to the parietal peritoneum to prevent leakage into the general peritoneal cavity.

Post-operative treatment should consist of the usual sedatives, glucose and insulin given frequently with a constant check on the blood sugar. In the post-operative care the internist should take an important part. The patient should be kept under observation for a period of years.

SURGERY OF THE PANCREAS IN DIABETES.

Of recent devolopement in this country is the experimental work of DeTakats in Chicago. He has shown that by either, complete severence of the tail of the pancreas from the body of the gland, or by ligating the body of the gland near the tail, with just enough pressure of the ligature to obstruct the duct of Wirsung, that a most interesting change takes place in the tail of the pancreas so separated from the body of the gland.

The changes noted are the production of fibrosis and disappearence of the excretory glandular part with an increase in the islet tissue, this increase in islet tissue resulting in an increased tolerance for carbohydrates. Where the tail has been entirely severed from the body of the pancreas the productive fibrosis increases to such an extent that not only is the excretory glandular part destroyed but also the islet tissue. However, when the body near the tail is ligated with only sufficient pressure to obstruct the duct, this marked fibrosis does not occur. The excretory glandular part becomes destroyed bu the islet tissue increases in amount as well as in activity.

This procedure has been performed on a case of severe juvenile diabetes with most encouraging results. The carbohydrate tolerance increased and the patient's insulin is gradually reduced. I believe that this procedure has great promise in selected cases. However, we must wait patiently for accurate observations extending over a period of years before the procedure can be expected as a standard procedure. DeTakats deserves a great deal of credit for blazing the trail in an unexplored surgical field.

BIBLIOGRAPHY.

1. McMurrich: The Development of the Human Body: P. Blackstone and Co., 1913.

2. Cunningham: Textbook of Anatomy, Revised Edition (4th), William Wood and Co.

3. Moynihan, Sir Berkley: Abddominal Operations, W. B. Saunders and Sons, 1926, volume 11. Acute pancreatitis, Ann. Surg., 81:132, 1925.

4. Maugeret, R.: Cholecyste-Pancreatite, Thesis, Paris, G. Steinheil, 1908 (Quoted by Graham).

5. Braithwaite, L. R.: The flow of lymph from the ileocaecal angle and its possible bearing on the cause of duodenal and gastric ulcer, Brit. Surg. Jour., 11:7, 1923.

6. Wilkie, D. P.: Brit. Jour. Surg., 15:450, 1928.

7. Deaver and Pfieffer: Tr. Amer. Surg. Assn., 31, 1913. Tr. Amer. Surg. Assn., 1922 (Quoted by Kaufman), Ann. Surg., 59, 1914.

8. Franke: Deutsch. Ztsch. f. Chir., Sept., 1911 (Quoted by Kaufman).

9. Bartels: (Quoted by Kaufman).

10. Graham and Peterman: Further observations on lymphatic origin of cholecystitis, choledochitis and associated pancreatitis, Arch. Surg., 4:23, 1922.

11. Kaufman, M.: An experimental study of the lymphatic theory of pancreatis, Surg., Gyn. and Obs., 44:15, 1927.

12. Ordway: Jour. Med. Research, 16, (Oct.) 1909 (Quoted by Kaufman).

13. Lancereaux: (Quoted by Mann and Giordano).

14. Opie, E. L.: The etiology of acute hemorrhagic pancreatitis, Bull. Johns Hopkins Hosp., 12:182-188, 1901.

15. Flexner, Simon: Experimental pancreatitis, Johns Hopkins Report, 9:743, 1900.

16. Polya: (Quoted by Moynihan).

17. Nordman: (Quoted by Moynihan).

18. Archibald and Gibbons: Ann. Surg., 74:426, 1921.

19. Egdahl, A. A.: Review of 105 reported cases of acute pancreatitis, with special reference to etiology, with a report of two cases, Bull. Johns Hopkins Hosp., 18:130-136, 1907.

20. Mann and Giordano: The bile factor in pancreatitis, Arch. Surg., 6, 1923.

21. Ochsner, A.: Drainage of abscess of pancreas, Ann. Surg., 74:434, 1921.

22. Hunt, Ernest L.: Pancreatitis, acute and subacute; some special problems in postoperative care, New Eng. Jour. Med., 198:610, 1928.

23. Guleke: (Quoted by DeTakats).

24. Unger: (Quoted by DeTakats).

25. Hess: (Quoted by DeTakats).

26. DeTakats, G.: General considerations and review of the literature, Arch. Surg., 19:771, 1929.

27. Watkins, Royal: Acute pancreatitis, New Eng. Jour. Med., 198:605, 1928.

28. Jones, T.: (Quoted by Moynihan).

29. Warren and Dermis: Surg. Clinics of N. Amer., 3:1319, 1923.

30. Judd, E. Starr: Mayo Collected Papers, 1921.

31. Ewing: Neoplastic Disease, Third Edition, Saunders, 1928.

32. Judd, E. Starr and Parker, B. R.: Biliary intestinal anastomosis for obstructive jaundice; analysis of 137 consecutive cases, Arch. Surg., 17:1, 1928.

33. Elkin, D. C.: Cancer of the pancreas and bile passages, Jour. Med. Assn. of Ga., 15:173-174, 1928.

34. Schmeiden and Sebening: A review of abdominal surgery. Nagel, G. W., Judd, E. Starr. Parker, B. R., Bueermann, W. H. and Peiper, H.: Arch. Surg., 19:537, 1929.

35. Jones, D. Fiske: Surg. Clinic of North Amer., 11:1125, 1922.

36. Cabot, Richard C.: Boston Med. and Surg. Jour., 194:207, 1926.

37. Cullen and Friedenwald: Acute and chronic pancreatitis, Arch. Surg., 15:1, 1927.

38. Bowler, John P.: Some aspects of pancreatitis, New Eng. Jour. Med., 198:568, 1928.

39. Archibald, E.: Acute edema of the pancreas, 90:803, 1929.

40. Holden and Moran: (Quoted by Bowler).

CURRENT PROBLEMS OF MEDICAL EDUCATION.—All true education is self-education. There is a distinct shift in many medical schools now toward placing greater responsibility on the student for his own training in an effort to emphasize learning by the student in distinction to teaching by the faculty. It is in the direction of individualizing instruction and providing opportunities for learning, for self-development and for independent work. This new emphasis is illustrated by the breaking down of the rigid class system; the discontinuance of uniform time and course schedules; the introduction of small teaching sections; personal contacts between students and instructors; provision for reasonable free time for reading, individual work and leisure; a reduction in the amount of lecturing, and the providing of opportunities for students who desire and are competent to do independent work. The aim is to develop minds capable of finding and appraising evidence and drawing conclusions based on sound reasoning which will provide a permanent intellectual equipment, resourcefulness and sound habits as well as methods of study that will permit the student to continue his own self-education throughout his entire professional life, a continuing education which has already been emphasized as the most important factor in providing an adequate medical service for the community.—Rappleye, W. C.: J. A. M. A., 94:916, 1930.

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YELLOW FEVER.

It may be recalled that a few years ago a leptospira was isolated by Noguchi from the blood of patients in Central and South America presumably ill with yellow fever, which he named Leptospira icteroides and which he believed was the cause of the disease. Several years later (1925) the West African Yellow Fever Commission undertook to investigate the disease as it occurred in Africa. By 1928, as a result of the studies of this Commission and of other independent investigators, considerable doubt hazed the whole question of the etiology of yellow fever. Noguchi's work was not confirmed and these problems arose: first, was yellow fever the same in America as in Africa? second, was the disease studied by Noguchi really yellow fever? The answer to these questions seems to be satisfactorily answered in a recent article by Sawyer, Kitchen, Frobisher and Lloyd.* Extensive bacteriologic, immunologic and pathologic studies of humans who had had yellow fever and of monkeys given the disease were made by this group. They conclude from their investigations that yellow fever is the same whether occurring in the Old or New World; that cases of Weil's disease (leptospiral jaundice) were present among those cases diagnosed as yellow fever studied by Noguchi; that Leptospira icteroides is an incitant of a form of infectious jaundice, resembling closely yellow fever and that "Noguchi's discoveries become of the greatest significance in respect to the epidemiology and causation of yellow fever and of infectious jaundice, previously confused one with the other." In the future due attention must be paid, during supposed yellow fever outbreaks, to the two kinds of jaundice, the one due to yelow fever virus and transmitted by the mosquito, the other to leptospirae and not controllable by anti-mosquito measures.

*The Relationship of Yellow Fever of the Western Hemisphere to That of Africa and to Leptospiral Jaundice, J. Exper. Med., 51:493, 1930.

POST-GRADUATE TEACHING.

Under the auspices of the University of Minnesota, Department of Medicine, there will be held the first part of July a symposium on the Kidney in Health and Disease. The program that has been arranged for this meeting could hardly be improved upon. Undoubtedly the most outstanding

men in the world who have made special studies of the kidney will be present to present and discuss certain phases of their subject. There appears on the program the names of such men as Volhard, Frankford-on-the-Main, Germany, certainly the greatest and best known worker in the fields of renal disease; Richards who has made epochal contributions to the physiology of the glomerulus; Rehberg from Copenhagen; Rowntree from Mayo Clinic; Berglund, notable for his work on disturbed physiology of the kidney and more particularly changes in the blood chemistry; Hirschfelder; Longcope; besides other men who are interested in other phases of the subject and have made a special study of them.

This symposium is open to any medical practitioner in the United States without cost. Special arrangements have been made to accommodate visiting doctors in the dormitories of the University. Any individual interested and wishing greater amount of information concerning this meeting should write to Dr. H. Burgland, University Hospital, Minneapolis, Minnesota.

This symposium offered to the practitioners of medicine in the United States illustrates very well indeed the growing tendency for medical schools and universities and medical societies to give to the practitioners of medicine special courses which will refresh and reburnish the minds of the hearers. It is an absolute impossibility for a busy practitioner of medicine to keep up to date on advancing phases of medicine. These courses offer the opportunity to him of getting and securing an immense amount of information, reliable and authentic. It is being very generally recognized by educators that these review weeks, symposium weeks, and so on have a distinct place in medicinal education. They are valuable and for the most part are eagerly sought after and grasped by the practitioner.

DECEPTIVE ADVERTISEMENTS.

Under the guise of a title suggesting a scientific publication and camouflaged by an appearance which would make the physicians unaware of the type of publication, the notorious Koch of Detroit has flooded this section of the country with a pseudo-medical journal which is just about the extreme limit of audacity. It may be that an occasional physician of repute is not aware of the so-called cancer cure elaborated by this man Koch. If not, it might be well to warn him that the Koch laboratories are putting out a secret nostrum spoken of as an synthetic toxin for the cure of cancer and its allied diseases, which has been totally and repeatedly refuted by scientists and thinking physicians. The man himself has been expelled from the medical organizations of organized medicine. Descending from what he originally claimed was a scientific preparation he has come out boldly with many schemes to advertise a product of which the claims are unfounded. The rewards that come to he who is sufficiently unethical to advertise, and low enough to advertise a secret substance, may be inferred from the fact that 150,000 copies of this journal are published, according to the notice made on page 23. It pays to advertise; assumedly the expense of printing and mailing this journal must be enormous, but it must pay or the astute business men of the Koch concern would not countenance such expenditures.

Physicians throughout Louisiana and Mississippi who are not familiar with Koch's efforts to achieve fortune through the suffering of others are warned of the character of his preparation. The majority of those who do know of this "cancer cure" can not help but feel insulted that their intelligence and integrity should be considered so low that they would pay any attention to such a journal as has been recently sent through the mails. Do not consign it to the waste paper basket, throw it in the garbage can.

HOSPITAL STAFF TRANSACTIONS

VICKSKBUFG SANITARIUM AND CRAWFORD STREET HOSPITAL.

Staff Meeting, April 10, 1930.

Abstract: A Few Remarks on Spinal Anaesthesia in Abdominal Surgery.—Dr. J. A. K. Birchett, Jr.

Subarachnoid or spinal anaesthesia was first described by Corning, the pioneer of local anaethesia, in 1885. His work consisted of two examples, one performed on the dog, the other on a human being complaining of spinal irritation. He injected cocain between the spinous processes thence to be carried to the cord by way of the veins which run to the venous plexus of the cord. Later in 1888 he treated and gave relief to four patients with pain from spinal cord lesions. At that time he did not realize or emphasize its use as a surgical anaesthetic. In 1894 he published a monograph on "Irrigation of the Cauda Equine with Medical Fluids." In this he advised that cocain be injected directly into the lumbar sac by puncturing between the first and second lumbar vertebrae. Interesting as this fact is, especially in its relationship to present day surgery and anaesthesia, it attracted slight attention and was soon lost until Bier in 1899 performed eight anaesthesias by the lumbar puncture route, one on himself, one on his assistant, and six on patients. The larger the dose of cocain, the higher was the level of anaesthesia noted. One extended as high as the umbilicus and on six, major operations could be done painlessly. Unfortunately the prompt disadvantage of violent headache lasting for days presented itself. The method did not meet with favor in Germany but in France and America it was used increasingly. Cocain having proved dangerous, the advent of stovain introduced by Fourneau in 1904 and the use of the suprarenal preparations of the day, caused the method to be recommended for general clinical use.

The spinal fluid which is the medium into which the anaesthetic solution is introduced, is worthy of review. The specific gravity is of importance in reference to the fluid to be introduced. The amount of spinal fluid is from fifty to one hundred and fifty cc. and the spinal pressure is sixty to one hundred mm. of mercury with the patient in the recumbent position. Changes in posture affect the fluid position. In the sitting posture the fluid reaches the upper dorsal region of the cord. In slight Trendelenburg position, not a drop can be obtained on puncture, as the fluid gravitates away from the lumbar region. If coloring matter is introduced in the lumbar region and the patient is then placed in high Trendelenburg posture, the coloring matter is noted at the base of the brain. This is of importance for if the anaesthetic solution mixes thoroughly with the fluid in too high a concentration there is danger of anaesthesia of the brain structures and the higher centers. To overcome this Barker employs the use of fluid heavier than the spinal fluid or by adding a substance with greater viscosity. This will, with difficulty mix with the spinal fluid and will stay in the most dependent part. In this way the position and heighth of anaesthesia can be controlled by changing the position of the patient.

Of the anaesthetic solutions employed, cocain was first used and abandoned due to toxicity. Tropococain, discovered in 1891, was first used by Chadbourne in Boston. This is readily soluble, is non-irritating and is one-third as toxic as cocain. It cannot be used with adrenalin. Stovain, previously mentioned, is readily soluble in water, may be boiled, has antiseptic properties, but its action is fleeting and it will cause gangrene if allowed to come in contact with tissues. Its action is rapid and muscles in the anaesthetic area becomes rapidly paralyzed. Novocain, a German preparation, was developed in 1905 and used extensively. It is readily soluble, can be heated to 120°C., but should not be boiled with adrenalin as it is decomposed if boiled too long. It is one-seventh as toxic as cocain, has no effect on the heart, in ordinary doses does not lower blood pressure, and is non-irritating to the tissues. Novocain affects the motor nerves less than stovain but in novocain the danger of respiratory paralysis is much less. A five per cent isotonic solution is used,—two cc. for operation on the genital organs, two and one-half cc. for operation on the legs and groins, and two and one-half to three cc. for laparotomy. In preparing this anaesthetic, some technicians dissolve tablets of 0.05 gms. of novocain in spinal fluid and reinject this mixture.

As to complications and sequelae met with in spinal anasthesia, during the operation the patient may be seized with nausea, retching and vomiting and show evidence of extreme shock. Severe headache and even cessation of respiration may occur. A main group of symptoms is spoken of as "meningism," which is a syndrome arising from a septic irritation of the meninges and due to leakage through the arachnoid puncture after the needle is withdrawn. Its train of symptoms are intense headache, dizziness, nausea, vomiting and pain in the back.

Headache is present usually after novocain and stovain; never as rule after tropocain. In one thousand cases in which spinal anaesthesia was used in the Jackson Clinic, the success of the procedure was in part due to the discovery of

ephedrine. Given before spinal injection, all shock is comparatively eliminated because the vascular tone is maintained. Under spinocain satisfactory anaesthesia was not always obtained, and in twenty-five per cent anaesthesia had to be completed with inhalation. Spinal anaesthesia has been used in all major operations below the diaphragm and in cystoscopy. Some have operated on the thyroid, mastoid, and cervical glands.

Labat states that spinal anaesthesia can not be used in operations for ruptured viscera, such as perforated ulcer and appendix, as it stimulates peristalsis and spreads infection. In the Jackson clinic it was found that the viscera were quieted and relaxed, also that the relaxation is so complete that it is not necessary to use packs. Postoperative complications such a pneumonia and phlebitis are unknown and surgery is facilitated twenty-five per cent because of the relaxation obtained.

No contra indications are known except cerebral neoplasm. Hypotension is controlled by ephedrine. Headache is practically eliminated by the use of a twenty-two gauge needle for the puncture. Ephedrine offsets nausea and vomiting due to fall in blood pressure. Barbital will control the danger of novocain reaction.

Technic: Give 1/3 grain of pantopon and 1/150 grain of scopolomine. Give ephedrine. Wait five minutes after ephedrine before injecting the novocain solution.

During the past month in this hospital spinal anaesthesia has been successfully used in operations for carcinoma of the omentum, repair of laceration of urethra, hemorrhoidectomy, postoperative ventral herni, caesarian section salpingectomy, oophorectomy, and appendectomy, hysterectomy with double salpingectomy and oophorectomy, supra public prostatectomy, hyterectomy (3 cases), salpingectomy and appendectomy, hysterectomy, double salpingectomy and oophorectomy, and peritonitis with gangrenous appendix.

Abstract: Carcinoma of the Appendix.—Dr. G. M. Street.

Patient: White female, aged 17 years, single, student. Admitted to hospital March 3, 1930.

Chief Complaint: Severe pain in right lower quadrant.

Present Illness: Began two weeks ago with severe pain in right lower quadrant; some nausea; no vomiting. Began at 5 a. m. and pain waked patient up. No diarrhea; severe pain lasted one hour and then has been intermittent ever since. Constipated.

Past History: Has had influenza, whooping cough, measles; rheumatism at the age of three. Tonsils and adenoids removed at age of nine; no other operations. Suffers with frequent severe headaches, both frontal and occipital. No history of pyelitis. Present weight under average about eight pounds. Menstrual periods have always been irregular and too infrequent. Has suffered with backache most of the time since a severe fall at the age of 11 years. Some bladder frequency and burning at times. Has had hemorrhoids which are rarely painful and ever bleed. Several previous attacks similar to the present one, the first about one year ago.

Family History: One sister has had a goitre which has been removed. No history of tuberculosis or cancer in the family. Mother and father, one brother and two sisters living and well.

Physical Examination: Nothing remarkable except a very sharply localized point of tenderness over McBurney's point and persistently in exactly the same location. No abdominal masses; Pelvic examination negative. Leukocyte count, 17,300; differential leukocyte count, small lymphocytes, 21 per cent; large lymphocytes, 1 per cent; large monouclears, 9 per cent; polymorphonuclear neutrophils 67 per cent (40 immature forms); polymorphonuclear basophils 2 per cent. No malaria found. Wassermann and Kahn tests negative. Urine not remarkable. Roentgen-ray examination of the pelvis and lumbar and dorsal spine, anterior posterior and lateral, negative.

Operation: McBurney incision; appendix in retrocecal position but easily delivered with cecum and gentle traction on base of appendix. If appendix was adherent, it was very slightly so; no dense adhesions. The distal 2/3 of the appendix was very hard and thickened with marked scarring and white solid areas showing through the peritoneal covering; appeared to be tubercles or caseous areas in the wall. Diagnosis at operation was probably tuberculosis. Mesentery and the area around where appendix was located were searched for glands or other pathology and none were evident. Right tube and ovary picked up and inspected and found normal. Wound was closed without drainage. Patient made an uneventful recovery.

Microscopic examination of appendix showed adeno-carcinoma (group III).

Abstract: Liver Abscess.—Dr. A. Street.

Patient: White male, aged 47 years, carpenter. Admitted to hospital March 30, 1930.

Chief Complaint: General aching, fever, nausea, anorexia and cough without expectoration; onset three days previously.

Previous History: Not remarkable except for attack of bloody diarrhea one month ago, lasting for ten days. The patient has resided in the United States all of his life.

Family History: Not remarkable.

Examination: Thorax shows dullness, bronchial breathing and moist rales at right base posteriorly. No other remarkable findings. Leukocyte count 22,000; neutrophiles, 85 per cent; urine not remarkable.

Course: Temperature 99°F. to 102°F. for one week. Then had sudden right upper quadrant pain, followed by tympanites, extreme weakness, rapid pulse and evidence of severe shock. Abdominal rigidity and tenderness, most marked in the right upper quadrant. The picture was that of acute peritonitis. As patient was in no condition for exploratory operation, palliative treatment was given,—ice to abdomen, turpentine stupes, colon irrigations for tympanites, no food by mouth, stomach kept empty with Jutte tube, and glucose intravenously.

Condition gradually improved and on March 24 there was tenderness and slight edema of lower right lateral chest. Exploratory needle introduced into the eighth interspace yielded chocolate colored thick pus. A trocar was introduced into this abscess through the same space and a pezzar catheter was introduced through the canula.

The abscess contained about one quart of chocolate colored pus. Ten cc. of Dakin solution were injected every three hours through the catheter. One grain of emetine was given intramuscularly each day for seven days.

Improvement was prompt and has continued. The patient now has no symptoms and the cavity is down to a capacity of one ounce. The temperature has been normal since March 28.

The chocolate colored fluid was sterile and no amebae could be found in it. However, in view of the history and absence of bacteria from the abscess contents, the diagnosis of amebic abscess seems justifiable.

CHAMBERLAIN-RICE HOSPITAL.

Dr. J. G. Logan reports for the Staff of the Chamberlain-Rice Hospital, Natchez, at its April Staff Meeting. Interesting cases:

1. White female, aged 8 years. Extensive cranial fracture. One linear fracture from vertex to right orbit; another from occipital region to right temporal region. Hemorrhage from right ear and nose. Marked evidence of intracranial pressure. Trephine and decompression over right temporal area under amytal anesthesia. Uneventful recovery.—J. C. Rice, M. D.

2. White female, aged 25 years. Marked pulmonary tuberculosis. History of recurrent left kidney colic for past several years. Severe left kidney colic for five days prior to admission. Roentgen-ray revealed a large stone in the pelvis of the left kidney. Ureteral catheterization showed good function of both kidneys and no evidence of tuberculosis of the kidney. Under amytal anaesthesia, nephrolithotomy was done and convalescence was uneventful. Patient has since entered the Mississippi State Sanitorium for pulmonary treatment.—J. F. Chamberlain, M. D.

3. Mr. C. W., aged 49 years, white, presented himself for diagnosis on acount of difficult breathing and loss of weight.

Past History—Patient had enjoyed good health up until about eight years ago when he awoke one morning and found that he had lost his voice. He suffered no pain and dyspnea at that time. He reports that he consulted a physician who prescribed for him and advised him that it was a paralysis of the vocal cord. He later consulted another specialist who advised him that it was in all probability due to a luetic process of many years standing. He states that he has continued about the same until six weeks before admission to the hospital when he almost died from trying to repress a cough in church. He states at that time he was breathless for some time. He has lost twenty-five to thirty pounds in weight.

Family History—Essentially negative.

Present History—He says that he came to the hospital on account of the fear that he would die unless something was done.

Physical Examination—Patient is an adult white male apparently fifty years of age, suffering with considerable dyspnea and unable to speak above a whisper. General physical and laboratory findings were essentially negative. The mouth showed no teeth present and a hypersentive throat and pharyngeal refiex. Under a local anesthetic and with the aid of the directoscope a section of the tumor mass extending up between the vocal cords was removed for laboratory diagnosis. The laboratory report was that the growth was benign. This was done on March 31, 1930. On April 2, a preliminary tracheotomy was done to relieve the dyspnea. On April 4, the tumor was removed by means of a laryngo-fissure and the pedicle was

fulgurated with diathermy. There was no packing and on April 6 the trachectomy tube was removed and the wound packed with gauze. Patient thus far has enjoyed an uneventful recovery. The gross pathological findings were those of a large benign papilloma.—Raymond T. Smith, M. D.

TRANSACTIONS OF THE CHARITY HOSPITAL SURGICAL STAFF.

The regular monthly meeting of the Staff was held on March 19, 1930.

Dr. Reginald A. Cutting presented a most interesting illustrated lecture on acidosis and alkalosis. Although his time was limited, he carefully explained the important points of each, and the importance of making a differential diagnosis, admitting that it was almost impossible to make a diagnosis clinically, and that blood chemistry was needed to differentiate. He also stated that insulin should not be used when administering glucose intravenously, because, theoretically at least, it seemed to be against certain physiological principles.

In discussing Dr. Cutting's remarks, Dr. E. L. King explained that at present there exists two schools in regard to this matter. One believes that insulin should not be given when glucose is administered, and the other believes that insulin is a necessary adjunct when glucose is given intravenously. In his own experience he pointed out that with cases of pernicious vomiting due to pregnancy, marked improvement was apparent when insulin was used with glucose intravenously, in contrast to only a slight improvement when insulin was not used with the intravenous glucose.

The next presentation was a brief discussion on the use of stock vaccines in the treatment of carbuncles by Dr. Shirley Lyons. Dr. Lyons presented a resumé of thirty cases treated by him, which were all benefited, and the period of disability shortened to an average of eighteen days. This presentation was thoroughly discussed by Drs. A. C. King, H. B. Gessner, F. L. Cato, and James Rives. It was brought out that the initial dose, as well as the subsequent doses, were to be very large if any benefit was to be derived.

The next matter of interest was the presentation of surgical deaths. The first case was that of a colored female who died from an extensive carcinoma of the cervix. The discussion which followed concentrated on whether or not radium should be used in very advanced cases of this type. The second case was that of a young negro male, 12 years of age, who died of an extensive lymphosarcomatous growth involving the intestines and mesentery, with metastatic foci in the liver and lungs.

FRANK L. LORIA, M. D.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY.

CALENDAR.

May 2—Pathological Conference, Hotel Dieu, 10-11 A. M.
May 2—Physiology Seminar, Tulane University, 5 P. M.
May 5—Eye, Ear, Nose and Throat Hospital Staff, 8 P. M.
May 9—Pathological Conference, Hotel Dieu, 10-11 A. M.
May 9—Physiology Seminar, Tulane University, 5 P. M.
May 9—French Hospital Staff, 8 P. M.
May 9—Medical Reserve Corps Branch School, 8 P. M.
May 12—**Orleans Parish Medical Society,** 8 P. M.
May 13—Baptist Hospital Staff, 8 P. M.
May 14—Touro Infirmary Staff, 8 P. M.
May 15—I. C. R. R. Hospital Staff, 12 N.
May 15—Eye, Ear, Nose and Throat Club, 8 P. M.
May 16—Pathological Conference, Hotel Dieu, 10-11 A. M.
May 16—Physiology Seminar, Tulane University, 5 P. M.
May 19—Hotel Dieu Staff, 8 P. M.
May 20—Charity Hospital Medical Staff, Jung Hotel, 12 Noon.
May 21—Charity Hospital Surgical Staff, 8 P. M.
May 23—Pathological Conference, Hotel Dieu, 10-11 A. M.
May 26—**Orleans Parish Medical Society,** 8 P. M.
May 30—Pathological Conference, Hotel Dieu, 10-11 A. M.

During the month of April the Society held one meeting. The regular quarterly executive meeting scheduled for April 14 was postponed to be held jointly with the special meeting on Saturday, April 19.

Dr. Charles L. Scudder, of Boston, Chairman of the Committee on Fractures of the American College of Surgeons, was the guest of the Society at the special meeting held Saturday, April 19. Dr. Scudder gave a very interesting talk on Industry and Fractures. Because of the season of the year this meeting was very poorly atended.

The regular meeting scheduled for April 28 was dispensed with because of confliction with the meeting of the Louisiana State Medical Society in Shreveport.

A special meeting of the Board of Directors was called on March 31 to consider the proposed drive for the Flint-Goodridge Hospital and Medical Center. At this meeting the following resolution was adopted:

"Recognizing the importance to the Community of such a health center and hospital for Negroes as is proposed in connection with the merger from which is to arise Dillard University, and a new Flint-Goodridge Hospital,

Be it resolved, That the Orleans Parish Medical Society heartily approves and endorses this project, and the campaign to collect the necessary funds to bring it to realization.

Be it further resolved, That a copy of this resolution be transmitted to the Committee in charge of the Flint-Goodridge Campaign."

Dr. Aaron J. White was elected to Active Membership.

We regret to learn of the resignation of Dr. Gayle Aiken.

The History being written by Dr. Fossier is finished. The members having their pictures in this book are requested to send in their checks to defray the expense of photo-engraving and the cost of the History.

TREASURER'S REPORT.

Actual Book Balance, Feb. 28, 1930	$3,004.21
Receipts	$3,214.50
	$6,218.71
Expenditures	$2,507.94
Actual Book Balance: March 31, 1930	$3,710.77

LIBRARIAN'S REPORT.

During March, 1930, 104 books have been added to the Library. Of these 44 were received by binding, 18 from the New Orleans Medical and Surgical Journal, 2 by purchase and 40 by gift. New titles of recent date are listed below.

The library is called upon more and more, to furnish material and information for particular cases in practice. We feel that this is the most worthy purpose of our medical library, and every effort is made to furnish the best articles and texts on the various subjects.

We have interested a young lady, recommended by Newcomb Art School in medical drawing, with the idea of having someone to whom we could refer doctors needing work of this kind. Another young lady has interested herself, at Miss Marshall's suggestion, in assisting doctors in the writing of medical papers. Both are doing most satisfactory work. Where translations are needed we refer the physicians to translators. In every way we try to make the information desired, available.

NEW BOOKS—MARCH.

Meagher—Study of Masturbation. 1929.
Harper—Clinical Obstetrics. 1930.
Pool—Surgery at the New York Hospital. 1930.
Christian—Physiology for Nurses. 1929.
Collins—Insomnia and How to Combat It. 1930.
Crooks—Aids to Orthopedic Surgery. 1929.
Pearce—Orthopedic Nursing. 1930.
McCollum—Newer Knowledge of Nutrition. 1929.
Horsley—Research and Medical Progress. 1929.
Rhinehart—Roentgenological Technique. 1930.
Schafer—Essentials of Histology. 1929.
Nissen—Practical Massage. 1929.
Campbell—Orthopedic Surgery. 1930.
Piney—Clinical Atlas of Blood Diseases. 1930.
Wright—Applied physiology. 1929.
Fakata—Funktion der weib-lichen Geschlechtsorgane. 1930.
Weinmann—Law Concerning Dead Bodies. 1929.
Pusey—Medicine as a Career. 1930.

H. THEODORE SIMON, M. D.,
Secretary.

LOUISIANA STATE MEDICAL SOCIETY NEWS

H. Theodore Simon, M. D., Associate Editor.

REGIONAL CONFERENCE ON SOCIAL HYGIENE.

The Southern States Regional Conference and Social Hygiene Institute will be held in New Orleans, May 23-27, under the auspices of the Louisiana State Board of Health and the New Orleans Council of Social Agencies. It is expected that health officers, physicians, public health and clinic and school nurses, educators, social workers, parents, club women, church leaders and others interested in social hygiene will be in attendance.

May 23-24 round table discussions of technical social hygiene problems have been arranged. There will be two groups—one for doctors, nurses and social workers primarily interested in the medical aspects of social hygiene; such problems as the treatment of syphilis in pregnancy, the management and control of clinics and social case work will be considered. The second group, composed of educators, Parent Teachers Association workers, clubs and church leaders, and others interested in the educational aspects of social hygiene, will discuss such problems as the method and material of sex education, child questions and their answers, the part of religious workers in sex education.

On Sunday, May 25, two meetings, one of which has been especially arranged to bring together Negro leaders, have been arranged for the general public.

The Regional Conference proper will open on the morning of May 26 and will continue through the next day. The program will include addresses by national hygiene authorities on the subject:

"The Cost of Syphilis in Life and Wealth."

"What Social Hygiene Means to the Community."

"The Protection of Youth from Moral Hazards."

"Congenital Syphilis Can and Must Be Prevented."

"The Part of the Nurse in Social Hygiene."

"Clinics for Venereal Diseases. Why We Need Them. How to Run Them."

"The Parent and Sex Instruction of Children."

Among the speakers will be Dr. Lloyd Thompson, Father Alphonse Schwitalla, Dr. O. C. Wenger, Dr. James R. McCord, Dr. E. L. Swan, Dr. Walter Clarke, Miss Edna Moore and Miss Henrietta Additon.

AMERICAN SOCIAL HYGIENE ASSOCIATION,
370 Seventh Avenue, New York, N. Y.

INVITATION TO CONFERENCE ON SOCIAL HYGIENE.

A copy of a letter sent to the physicians of New Orleans is published below. This same invitation is extended to the physicians of Mississippi and Louisiana. Only the cost of sending so many letters has prevented the sending out of individual invitations throughout the two states.

American Social Hygiene Headquarters,
302 Audubon Building.

Dear Dr. Blank,

You are cordially invited to attend the Southern States Social Hygiene Conference, May 26-17, to be held at the Jung Hotel, under the joint auspices of the Louisiana State Board of Health and the Central Council of Social Agencies.

At this Conference, a distinguished list of speakers from out of town will be on the program. I feel that of particular interest to the physicians of New Orleans will be the special institutes held the evenings of May 23 and 24 at 7:45 P. M., at the Jung Hotel. These are closed meetings, open to certain small groups only, requiring a registration fee of $2.00, and are limited to one hundred participants.

Group 2 of the institute is arranged primarily for doctors. The first night the meeting will be led by Dr. James R. McCord and will deal with the prevention of congenital syphilis. Dr. McCord, Professor of Obstetrics, at Emory University, is a special representative of the U. S. Children's Bureau and has been engaged in a study of this subject under the auspices of the Rockefeller Foundation. The next evening's program will be conducted by Dr. O. C. Wenger, U. S. Public Health Service, and will take up the subject of clinics for the treatment of syphilis and gonococcal infections. Dr. Wenger is now demonstrating a model clinic under the direction of the U. S. Public Health Service. Dr. Walter Clarke, Medical Director of the American Social Hygiene Association, will also participate in this program.

A registration blank is enclosed which you are requested to fill out and return at your earliest convenience to American Social Hygiene Headquarters, 302 Audubon Building, New Orleans.

ST. TAMMANY PARISH MEDICAL SOCIETY.

The monthly meeting of the Society was held Friday evening, April 4, in Slidell, with the following physicians present: Doctors Gautreaux, Griffith, Singleton, Polk, Bulloch, Lawrence, Young, Paine, Roland Young Maylie, F. F. Young

and Dr. I. I. Lemann of New Orleans, invited guest.

Dr. Lemann was asked to address the meeting and delighted all present with an able discourse on "Angina and coronary occlusion." Dr. Lemann in his usual able manner, brought out all the late points of interest in connection with the condition, recited numerous cases that he had dealt with, that presented one or more of these features, making his talk very comprehensive. Dr. Lemann was given a standing vote of thanks for his instructive paper.

In response to a communication from Dr. P. T. Talbot, calling our attention to the action taken by the Rapides Parish Medical Society in reference to the Porter Bills, now pending in our National Congress, the society joined hands with the Rapides Parish Society in opposing said bills, and the Secretary was instructed to communicate with our Representatives in Congress appraising them of our opposition to said bills.

The Society had a lengthy round-table discussion of topics of the day, in general, then adjourned to meet next month in Mandeville.

H. D. BULLOCH, M. D.,
Secretary and Treasurer.

MEETING OF THE BI-PARISH MEDICAL SOCIETY.

The East and West Feliciana Bi-Parish Medical Society met in the East Louisiana State Hospital as the guest of Dr. Glenn J. Smith and staff. The Society instructed our Senators and Representatives in Congress to vote against and use their influence against the Porter Bills.

Drs. P. H. Jones, Jr., Cecil Lorio and O. H. Waltrip read instructive papers.

Members present were Drs. Smith, Waltrip, Miller, P. H. Jones, Sr., P. H. Jones, Jr., Cecil Lorio, L. F. Lorio, Williams, Sewell, Pittman, Lea, Rombard, Stenley, Fossae, Morgan, Shaw, Morris, Hargrove, McCaa, Cook, Irwin and Toler.

E. M. TOLER, M. D.,
Secretary-Treasurer.

FIFTEENTH ANNUAL CLINICAL SESSION OF THE AMERICAN COLLEGE OF PHYSICIANS.

The American College of Physicians will hold its Fifteenth Annual Clinical Session at Baltimore, Maryland, from March 23-27, inclusive, 1931. The Lord Baltimore Hotel will be headquarters.

Dr. Sidney R. Miller, Baltimore, as President, will have charge of the selection of the general scientific program. Dr. Maurice C. Pincoffs, of Baltimore, has been appointed by the Board of Regents as the General Chairman of the Session, and will make all local arrangements, including the making up of the program of clinics. Business details will be handled by the Executive Secretary, Mr. E. R. Loveland, from the College headquarters, 133-135 S. 36th Street, Philadelphia, Pa.

The attention of secretaries of various societies is called to the above dates, in the hope that their societies will select non-conflicting dates for their 1931 meetings.

NEWS AND COMMENT.

Dr. H. W. E. Walther, of New Orleans, addressed the Tri-State Medical Association, at Marshall, Texas, on March 21, 1930, his subject being: "The verumontanum in its relation to certain obscure sexual and urinary disturbances in the male."

Dr. H. Daspit, Dean of Graduate School of Medicine of the Tulane University of Louisiana, was a guest of the Ouachita Medical Society on Wednesday, April 16, 1930, at a special meeting held at Monroe, La., and addressed the group on "Paralyses resulting from jamaica ginger."

Assistant Surgeon General R. C. Williams, U. S. P. H. S., has been directed to proceed from Washington, D. C., to points in Tennessee, Alabama, Mississippi, Louisiana, Texas, Arknsas, and Kansas, and return, for the purpose of conferring with State and local health authorities relative to the reporting of measures for the prevention of smallpox and typhoid fever.

Dr. Lucien LeDoux was a guest of the Third District Medical Society at their last meeting. He read a paper on "The relationship of emotionalism to ovarian disturbances."

Beginning this year the American Association for the Study of Goitre will award a cash prize of $300 annually for the best original thesis dealing with some phase of the goiter problem. Theses should be submitted by June 1, to Dr. Walter M. Simpson, Chairman of the Essay Committee, Miami Valley Hospital, Dayton, Ohio. The award will be given immediately following the coming meeting of the Association which is to be held in Seattle, Washington, July 10-12, 1930.

CORRESPONDENCE.

Editor,

New Orleans Medical and Surgical Journal.

My Dear Sir:

I would appreciate your inserting a notice warning physicians and surgeons to guard carefully their prescription blanks. Recently several blanks were stolen from a desk in the waiting room of my office, presumably by a narcotic addict. He has endeavored to have filled rather crude prescriptions for morphine, which are written on these blanks and he may have succeeded in some instances. Several druggists have detected the forgeries and have refused, after telephoning me, to fill the prescriptions.

The moral is keep all prescription blanks carefully put away where unauthorized persons cannot get at them.

Yours truly,

March 31, 1930. E. L. KING.

NATIONAL HOSPITAL DAY.

National Hospital Day is now annually celebrated by thousands of hospitals in Canada and the United States, as well as other lands, because it provides the means of directing public attention to the great work which these institutions do in the humanitarian cause. The movement originated eight years ago and was an expression of the widely growing feeling that communities were not only entitled to information about hospital endeavor, but should in their turn afford more definite support to these projects which contribute to the health and happiness of citizens in general. The hospital today functions as a most important and essential utility and is closely related to the program of public health activities.

The particular object of the National Hospital Day movement is to encourage the public to visit a hospital on May 12 so that all may become acquainted with the methods of conducting these institutions, their causative, custodial, educational and research activities, and by so doing to dispel the old time fear of the hospital and to compel the conviction that it is the proper place to come to when one is ill.

THIRD GRADUATE FORTNIGHT.

The Third Annual Graduate Fortnight of the New York Academy of Medicine will be held from October 20-31, 1930. The general subject which has been chosen for this year is "Medical and surgical aspects of acute bacterial infections."

The program as arranged is in two parts—coordinated afternoon clinics to be held in ten important hospitals of the city, and evening meetings to be held at the Academy. An added feature of this year's Fortnight will be an exhibit of anatomical, bacteriological and pathological specimens and research material bearing upon the various aspects of the subject. Each of the hospitals co-operating in the Fortnight will present two afternoon clinical programs dealing with different phases of the general subject.

The program for the evening meetings to be held at the Academy includes discussion of:

Focal infections as a cause of disease.
Osteomyelitis and acute joint infections.
Acute infections of the genito-urinary tract.
Infections arising from tonsils and sinuses.
Infections of the middle ear.
Acute infections of the face and oral cavity.
Operative risks from infection.
Appendicitis.
Bacteremia.
Suppuration of lung and pleura.
Acute infections of the gall-bladder and biliary tract.
Infections of the skin and subcutaneous tissue.
Acute infections of the upper respiratory tract including influenza.
The pneumonias and other pneumococcus infections.
Bacteriophage as a treatment in medical and surgical acute bacterial infections.
Puerperal sepsis.
Immunity—general and local.
Serum therapy.
Vaccine and non-specific protein therapy.
Rheumatic fever.
Acute and sub-acute bacterial endocarditis.
Meningococcus infections including meningitis.

The list of speakers who have been invited to take part in the Fortnight includes prominent clinicians from many parts of the country who are recognized by authorities in their special lines of work. The profession generally is invited to attend. No fees will be charged for attendance at any of the clinics or meetings on the program. A complete program and registration blank for special clinics and demonstrations will be mailed on request. Write N. Y. Academy of Medicine, Fifth Avenue and 103rd St., New York.

RESOLUTIONS ON THE DEATH OF DR. MERRILL.

Whereas, Dr. Herman P. Merrill has been a member of the Bi-Parish Medical Society; and,

Whereas, Dr. Merrill has been respected and honored by all the members of this Society, and by all others who knew him, as a man of the highest moral and professional character; and,

Whereas, on Christmas morning, December 25, 1929, without warning, God in His almighty wisdom saw fit to take him from our midst;

Therefore, be it resolved, That by the death of Dr. Merrill the Bi-Parish Medical Society has suffered the loss of one of its most honored and beloved members, and offer their heartfelt sympathy to the surviving members of his family.

Be it further resolved, That a copy of this resolution be incorporated in the minutes of this Society, and a copy sent to the family of the deceased, and Secretary of Louisiana Medical Society.

J. W. LEA, M. D.,
C. S. MILLER, M. D.
O. H. WALTRIP, M. D.

PARISH MEDICAL SOCIETY OFFICERS.

The following Parish Medical Societies have elected officers for 1930:

Acadia Parish: President, Dr. A. R. Morgan, Crowley; Vice-President, Dr. L. L. Kahn, Rayne; Secretary-Treasurer, Dr. S. R. Henry, Crowley; Delegate, Dr. J. W. Faulk, Crowley; Alternate, Dr. J. D. Hunter, Rayne.

Bossier Parish: President, Dr. J. B. Hall, Alden Bridge; Secretary-Treasurer, Dr. C. M. Tucker, Haughton; Delegate, Dr. C. M. Tucker, Haughton; Alternate, Dr. J. B. Hall, Alden Bridge.

METAPHEN AS GERMICIDE AND SKIN DISINFECTANT—George W. Raiziss, Marie Severac and J. C. Moetsch, Philadelphia, report on the results of an extensive study of metaphen and many other antiseptics in blood serum and skin disinfection. The technic used to study the skin disinfection is in principle the same as that of Roberts, with certain modifications. They contend that only an antiseptic which passes the most rigid test is to be considered safe for use in all clinical cases in which a germicide is to be applied. They conclude, therefore, that, considered from this point of view, ethyl alcohol, which is used extensively, is disqualified as a skin disinfectant. A solution of hexylresorcinol 1:1,000 (S. T. 37) gave growths of bacteria in all tests. Acriflavine base was found to be efficient in 2 per cent concentration, resulting in sterilization in 80 per cent of cases. It is important to note that a 5 per cent solution of iodin is ineffective and a 7 per cent is effective in 88 per cent of cases. Two per cent mercurochrome-alcohol-acetone-water solution showed only 79 per cent efficiency. An aqueous solution of metaphen 1:2,500, or 0.04, produced sterilization in all of the fifty tests performed. Metaphen proved to be efficient in very high dilutions. Thus a 1:2,500 dilution of it, or 0.04 per cent, was found to produce 100 per cent sterilization, while 7 per cent iodin and 2 per cent mercurochrome sterilized at five minute intervals to the extent of 70 per cent and 50 per cent, respectively. This indicates that better results were obtained with metaphen in dilutions 175 and fifty times greater than those employed for iodin and mercurochrome, respectively.—J. A. M. A., April 19, 1930.

MISSISSIPPI STATE MEDICAL ASSOCIATION NEWS

L. S. Lippincott, M. D., Associate Editor

VICKSBURG, MISSISSIPPI.

LOCATION.

Vicksburg, Mississippi, located on the western border of the State of Mississippi, and approximately half way between the northern and southern boundary of the State, is built on the high bluffs and slopes overlooking the Mississippi and Yazoo Rivers which form the western boundary of the State.

The city, itself, is topographically and architecturally picturesque, and in common with Southern cities of ante-bellum days, exhibits a delightful blending of the old regime with the improvements of the present. It contains the largest and one of the most beautiful park-like National Cemteries, and what is the greatest attraction of all, the Vicksburg National Park.

HISTORY.

Vicksburg was established in 1791 by the Spaniards as Fort Nogales. It was later chartered as the city of Vicksburg in 1825. In 1861, when the flames of war spread over the country, Vicksburg was recognized by both the Federals and Confederates as an extremely advantageous point. Of such importance was the city of Vicksburg to both the North and South that General Grant said: "When Vicksburg fell, the fate of the Confederacy was sealed."

After the Civil War and during the reconstruction Vicksburg also played an important part, and rapidly grew to be one of the most important points along the entire Mississippi River.

VICKSBURG NATIONAL MILITARY PARK.

This park, constructed by the United States Government, is in commemoration of the campaign, siege and defense of Vicksburg, which campaign, siege and defense extended over a period from March 20 to July 4, 1863.

The general plan and scope of the park is such that it is intensely interesting and instructive place to visit. It contains 1323 acres, and practically includes the battle lines of the two opposing armies during the investment of Vicksburg and all the fighting ground, between them. The park is traversed by thirty-two miles of graveled roadways. The park contains 897 tablets, in appropriate locations, 128 mounted guns, and 468 memorials, monuments and markets.

THE NATIONAL CEMETERY.

The National Cemetery at Vicksburg, fronting the river and blending into the northern end of the Military Park, although none of it, was established in 1865. This is one of the most magnificent cemeteries ever dedicated to the interment of the dead soldiers of any nation. It contains the graves of 16,22 Union soldiers, who lost their lives in and around Vicksburg during the Civil War.

The cemetery is a remarkable piece of landscape engineering with delightful walks and drives, with ravines, terraces and plateaus, and with long avenues of trees, most Spanish oaks and magnolias, supplemented with tropical plants and flowers.

NATIONAL PARK GOLF CLUB.

The beautiful links of the eighteen-hole National Park Golf Club are available to visiting golfers. This golf course is one of the finest in the entire Southland and its rugged, natural hazards make the playing of this course a real joy to the golf devotee.

EXCELLENT FISHING.

Numerous lakes, the largest of which is Eagle Lake, offer the sportsman every opportunity to enjoy a morning's fishing. The size and gameness of the famous "Eagle Lake large mouth black bass" is known the country over. A visit to Vicksburg is not complete for the sportsman without a side trip to Eagle Lake.

BRIDGE ACROSS THE MISSISSIPPI RIVER.

One of the most interesting side trips, will be the one to the site of the new seven million dollar ($7,000,000.00) combination highway and rail bridge across the Mississippi River. This immense structure has been recently completed and open to traffic and is the only bridge across the river south of Memphis, Tennessee.

MISSISSIPPI STATE MEDICAL ASSOCIATION

Dr. T. M. Dye, Secretary of the Mississippi State Medical Association, has announced the preliminary program for the meeting of the Association in Vicksburg on May 13, 14, and 15, as given below. This program is subject to some changes:

GENERAL MEETING.

First Day.

Tuesday, May 13, 1930.

Sessions: 9:30 A. M. to 12 M.; 1:30 P. M. to 6:00 P. M.

Auditorium—Y. M. C. A. Building.

OPENING EXERCISES.

1. Call to order. President H. A. Gamble, Greenville.
2. Invocation. Rabbi Sol Kory, Vicksburg.
3. Report of Committee on Arrangements.

SECTION ON SURGERY.

H. R. Shands, Chairman, Jackson

1. Treatment of Acute Peritonitis. R. D. Kirk, Jr., Tupelo.
2. Amytal as an Anesthetic. C. T. Chamberlain, Natchez.
3. Controllable Spinal Anesthesia. J. C. Culley, Oxford.
4. Surgical Mortality: Its Interest to the General Practitioner. William Bartlett, St. Louis.
5. Plastic Surgery About the Orbit. A. G. Wilde, Jackson.
6. Carcinoma of the Rectum. W. E. Sistrunk, Dallas, Texas.
7. Carcinoma of the Stomach. A. Street, Vicksburg.
8. Fracture of the Femur. F. H. Hagaman, Jackson.
9. The Diagnosis and Treatment of Ruptured Duodenal Ulcer. Gilruth Darrington, Yazoo City.

EVENING SESSION.

Eight O'Clock

Tuesday, May 13, 1930.

Auditorium—Y. M. C. A. Building.

To which the Public is Cordially Invited.

1. Invocation. Rev. Gordon M. Reese, Vicksburg.
2. Addresses of Welcome—
   On Behalf of the City of Vicksburg. Mayor W. J. Hossley.
   On Behalf of Issaquena-Sharkey-Warren Medical Society, Laurence J. Clark, Vicksburg.
3. Response to Addresses of Welcome. R. Curtis Smith, Drew.
4. Annual Oration. J. Shelton Horsley, Richmond, Va.
5. President's Address. H. A. Gamble, Greenville.

Wednesday, May 14

9 A. M.

Section on Eye, Ear, Nose and Throat

C. A. McWilliams, Chairman, Gulfport

Chairman's Address, C. A. William, Gulfport.

1. Headache of Nasal Origin. H. R. Fairfax, Brookhaven.
2. Corneal Ulcers. W. L. Hughes, Jackson.
3. Tonsillectomy Hemorrhages. E. F. Howard, Vicksburg.
4. Protein Therapy in Ophthalmology. A. G. Wilde, Jackson.
5. Enucleation of the Eye with Glass Ball Implantation. E. C. Ellett, Memphis.
6. Some Observations on Sinus Diseases in Children. R. C. Lynch, New Orleans.
7. Gross and Microscopic Pathology in Chronic Maxillary Sinus Disease. Robin Harris, Jackson.
8. The Common Cold in Ear, Nose and Throat Practice. D. W. Hamrick, Corinth.

SECTION ON PUBLIC HEALTH AND HYGIENE.

Hardie R. Hays, Chairman, Jackson

1. The Control of Venereal Diseases in Mississippi With Competent Treatment by the General Practitioner. Hardie R. Hays, Jackson.
2. Flocculation Test for Syphilis; Its Value. T. W. Kemmerer, Jackson.
3. Undulant Fever. H. E. Hasseltine, U. S. Army.
4. Snake Bite. Dudley Jackson, San Antonio.
5. Hookworm Disease and Infestation. Mr. C. A. Parmalee, Jackson.
6. Full Time Health Work in Mississippi. Felix I. Underwood, Jackson.

Wednesday, May 14

3:00 P. M.

SECTION ON RADIOLOGGY.

C. C. Hightower, Chairman, Hattiesburg

1. The Roentgen Diagnosis of Pulmonary Tuberculosis with Especial Reference to Pathological Classification. Geo. P. Sims, Gulfport.

2. An Interesting Study in Roentgen Ray From Memory's Storehouse. H. G. McCormick, Laurel.

3. Roentgen Ray Diagnosis in Gall Bladder Disease. Geo. M. Street, Vicksburg.

4. Duodenal Ulcer in Childhood. W. F. Henderson, New Orleans.

Wednesday, May 14

2:00 P. M.

G. Y. Gillespie, Jr., Chairman, Greenwood

1. Precordial Pain. L. J. Clark, Vicksburg.

2. Upper Left Abdominal Tumor Mass in Children, and Some Points in Differential Diagnosis. S. L. Brister, Jr., Greenwood.

3. The Cardiac and Other Disabilities of Advancing Years. Jas. S. McLester, Birmingham.

4. Paranasel Sinus Disease in Children. J. D. Simmons, Gunnison.

5. Colitis. C. W. Patterson, Rosedale.

6. The Use of Whole Blood in Pediatric Work. N. C. Womack, Jackson.

7. Some Observations on Gastro-Intestinal Disease. J. H. Musser, New Orleans.

8. A New Foreign Protein Which May Be Used Without the Usual Reaction. J. W. Gray. Clarksdale.

9. Acidosis and Alkalosis. G. L. Arrington, Meridian.

10. Epidemic Cerebro-Spinal Meningitis. F. M. Acree, Greenville.

11. A General Consideration of the Treatment of Syphilis (Lantern Slides). T. E. Ross, Jr., Hattiesburg.

NORTH MISSISSIPPI SIX COUNTY MEDICAL SOCIETY.

A meeting of the North Mississippi Six County Medical Society was held at Oxford on April 16, beginning at 11 A. M. and continuing throughout the afternoon. The program as announced by Dr. A. H. Little, Secretary, was as follows:

1. Vomiting Problems seen in Children. Dr. Ralph Bowen, Memphis. Discussion opened by Dr. J. B. Bailey.

2. Some Remarks on Applied Anatomy. Dr. P. L. Mull, University. Discussion opened by Dr. H. O. Leonard.

3. The Relation of the Physician to his Medical Society. Dr. H. A. Gamble, Greenville.

4. Cystitis. Dr. T. A. Moore, Memphis. Discussion opened by Dr. G. A. Brown.

5. Total and Differential Leukocyte Counts in Diagnosis and Prognosis. Dr. Leon S. Lippincott, Vicksburg. Discussion opened by Dr. J. M. Anderson.

6. Osteomyelitis. Dr. J. A. K. Birchett, Jr., Vicksburg. Discussion opened by Dr. H. P. Boswell.

Lunch was served at noon and the business session of the Society followed the lunch.

STATE ASSOCIATION GOLF.

Dr. Edley H. Jones, Chairman of the Committee of the Issaquena-Sharkey-Warren Counties Medical Society on the entertainment of the Mississippi State Medical Association, has announced that all entries for the golf tournament must be turned in to the registration office by 12 o'clock noon, Tuesday, May 13, and that all score cards must be turned in at the same place by 12 o'clock, noon, Wednesday, May 14.

The tournament will be for eighteen holes and is open to all members and guests of the Association. No entrance fee will be charged. Suitable trophies will be awarded.

CENTRAL MEDICAL SOCIETY.

The regular monthly meeting of the Central Medical Society was held on March 18, with Dr. R. W. Hall, Jackson, President, presiding.

A Clinic from 7:30 to 8:00 P. M., included the following:

Dr. N. C. Womach, Jackson, presented two cases in which intracranial hemorrhage had occurred at birth. The first patient, a girl of four months, showed complete recovery with no history of illness since treatment at birth for hemorrhage. The second patient, a boy of fourteen months, showed complete recovery twenty-four hours after birth.

Dr. Ainsworth presented a case report of a patient showing jaundice following a seventh dose of neoarsphenamine, this dose being given during a rest period by a doctor unfamiliar with the case.

Dr. G. W. F. Rembert, Jackson, presented case reports of four patients with "ginger" paralysis, with history of onset and neurological findings.

For the Scientific Program following, Dr. W. R. Bethea, Memphis, read a very interesting paper on "The Observation and Treatment of Some Conditions of the Newborn." Dr. Bethea urged a routine Roentgen-ray examination of all newborn babies as a means of decreasing infant mortality; among the conditions found and successfully treated are tracheo-esophageal fistula, enlarged thymus, congenital atelectasis.

Dr. Henry Boswell, director of the State Tuberculosis Sanatorium, read a paper on "Tuberculosis in Children." This paper was a compilation of the work of the leading authorities of America and will be released to the general profession in the near future.

Dr. Frank Hagaman, Jackson, presented an excellent paper on "Tuberculosis of the Joints." He stressed the importance of holiotherapy and rest of the involved joints, using surgery only as a last resort.

This was the first quarterly meeting of the year and was one of the best in the history of the society.

JACKSON INFIRMARY.

At the regular monthly meeting of the Jackson Infirmary on March 12, Drs. P. R. Greaves, J. C. Walker, and O. Simmons were elected to membership on the staff. Dr. J. E. McDill resigned from the Roentgen-ray department to resume private practice.

DELTA MEDICAL SOCIETY.

Dr. J. W. Lucas, Moorhead, Councilor for the First District of the Mississippi State Medical Association, furnishes the following:

"The Delta Medical Society met in semi-annual session in Greenwood on April 9, with a good attendance, interesting papers and discussions. The following papers were presented:

1. Cerebro-spinal Meningitis. Dr. R. M. Donald, Moorhead. Discussed by Dr. R. E. Wilson, Greenville, and others.

2. Rectal Strictures. Dr. G. M. Owens, Shelby. Discussed by Dr. George McMaster Barnes, Belzoni, and Dr. H. A. Gamble, Greenville.

3. Acute Inversion of the Uterus. Dr. L. B. Otken, Greenwood. Discussed by Dr. J. F. Lucas, Greenville.

4. Acute Agranulocytic Angina. Dr. H. G. Rudner, Memphis. Discussed by Dr. J. J. Shea, Memphis.

"By request from the chair, Dr. H. A. Gamble, president of the State Medical Association, made a splendid speech on the 'Good of the Order.'

"Belzoni will be the host at the next semi-annual meeting of the Society.

"The banquet held at the Rose Community Building was an enjoyable occasion, attended by 125 doctors and visitors. The banquet was presided over by Dr. T. B. Holloman of Itta Bena in a fitting manner. A hearty welcome by the President of the Chamber of Commerce, H. L. DeLoach, was responded to in a pleasing manner by Dr. W. W. Lewis, President of the Society. Dr. Tracy H. Clark, Chicago, a guest of the Society, responded to 'Opportunities of the Doctor for Service to Others.' Honorable W. K. Clemens gave a splendid address on 'Service.' The Blackstone-Henmen quartet was featured in a number of entertaining songs. The occasion was a success, the pleasure ours."

A son was born to Dr. and Mrs. J. E. Furr, physician in charge of the Marks Hospital, Marks, on February 11, 1930. The name of the young man is James Edward Furr, Jr.

On January 1, 1930, the Tylertown Hospital changed ownership. Dr. O. B. Harvey moved to Hattiesburg and sold his interest in the hospital to Drs. B. L. Crawford and J. B. Davis. The hospital has been incorporated and Dr. Crawford is President and Dr. Davis, Secretary and Treasurer. The institution has been re-arranged and new equipment added.

ISSAQUENA-SHARKEY-WARREN COUNTIES MEDICAL SOCIETY

The Regular monthly meeting of the Issaquena-Sharkey-Warren Counties Medical Society was held at Vicksburg April 8, with twenty-one in

attendance. The program included the following papers and discussions:

1. The Simulation of Visceral Lesions by Neuralgia of the Nerves Supplying the Abdominal Wall.—Dr. Edwin Press Hall, Vicksburg. Discussed by Drs. J. A. K. Birchett, Jr., G. M. Street, R. H. Foster, H. H. Haralson, W. H. Scudder, A. Street, E. F. Howard, and H. A. Gamble. Dr. Hall closed.

2. My Experiences with Eclampsia in Country Practice.—Dr. H. S. Goodman, Cary.

Discussed by Drs. A. Street, H. H. Haralson, W. H. Scudder, S. W. Johnston, R. H. Foster, A. K. Barrier. Dr. Goodman closed.

The feature of the meeting was an address by Dr. Hugh A. Gamble, Greenville, President of the Mississippi State Medical Association. Dr. Gamble talked in an interesting and instructive way on the ideals, purposes, and duties of the Medical Profession and the County Medical Society to organized medicine. The address was much enjoyed by every one present.

The various committees of the society on entertainment of the State Medical Association at its meeting in Vicksburg in May reported and everything will be in readiness when the association meets.

The society endorsed and commended the work of the Mississippi State Board of Health of the County Health Department in sanitation of barber shops.

There will be no regular meeting of the Society in May because of the meeting of the State Medical Association. In June the Society will meet in Monroe in joint session with the Fifth District Medical Society.

DESOTO COUNTY MEDICAL SOCIETY.

Dr. L. L. Minor, Secretary of the Desoto County Medical Society reports as follows: "At our regular meeting on April 7, 1930, an interesting program was gone through with. A luncheon was held at the Spencer Hotel in honor of our oldest and most esteemed member, Dr. William S. Weissinger of Hernando. The doctor was a valiant soldier of the Confederacy under General N. B. Forrest. In 1868 he began in this country as a general practitioner. He is still hale and hearty, an honor to his profession and to his country.

"Dr. J. V. Wright read an interesting paper on our group insurance plan.

"Dr. L. H. Brevura, formerly of this county, now living at Dundee, reported several interesting cases.

"Drs. A. L. Emerson, W. J. Gillespie, H. A. Stewart and W. J. Weissinger are on the program for the August meeting.

"Our youngest member, Dr. C. Whitley Emerson, is our President for the ensuing year.

"We all expect to be on hand in Vicksburg next month. Vicksburg is unsurpassed as a convention city."

Acknowledgement is made of the receipt of a reprint, "The Bridging of Osseous Defects of the Forehead, Using Metal Models as Guides for the Shaping of Cartilage Transplants," by Leslie V. Rush, M. D.; J. Hack Rush, D. D. S., M. D., and H. Lowry Rush, M. D., Meridian, Mississippi. This interesting article was published in the American Journal of Surgery in December, 1929.

Acknowledgment is made of receipt of a reprint, "Congenital Hypertrophic Pyloric Stenosis," by Dr. A. B. Harvey, Hattiesburg. This article was published in the New Orleans Medical and Surgical Journal in February, 1930.

Dr. L. B. Hudson, Hattiesburg, has announced the installation of roentgen-ray equipment and radium in his offices and laboratories at Hall Avenue and Arledge Street.

The next meeting of the North East Mississippi Medical Society will be held at Houston in June.

RUSH'S INFIRMARY.

Dr. H. Lowry Rush, Secretary of the Staff of Rush's Infirmary, reports that the regular monthly meeting of the attending and visiting staffs was held on April 4, with twenty-four members in attendance. The following scientific program was presented:

1. The Diagnosis of a Failing Heart.—Dr. T. D. Bourdeaux.

Discussed by Drs. Hart, Gully, Tatum, Rush and Robinson.

2. Urological Problems Pertaining to General Practice.—Dr. J. T. Bailey.

Discussed by Drs. Rush, Googe and Tatum.

At the conclusion of the program a plate lunch was served.

EAST MISSISSIPPI MEDICAL SOCIETY.

Dr. R. Lowry Rush, Secretary, of the East Mississippi Medical Society, reports the program for the meeting of the Society on April 10, at Meridian, as follows:

1. Acidosis.—Dr. E. L. Richardson, Louisville.

Discussion opened by Drs. T. E. Jarvis and T. E. Royals.

2. Salpingitis and When to Operate.—Dr. M. L. Flynt, Newton.

Discussion opened by Drs. C. H. Harrison, Philadelphia, and K. T. Klein, Meridian.

3. A Consideration of Eye Injuries.—Dr. C. P. Mosby, Meridian.

Discussion opened by Drs. H. L. Arnold and G. W. Bounds, Meridian.

4. Scarlet Fever.—Dr. H. F. Tatum, Meridian.

Discussion opened by Drs. J. T. Googe and T. D. Bourdeaux, Meridian.

Dr. Raymond T. Smith recently entertained Dr. Carl H. Christopher, eye, ear, nose and throat specialist of Mercy Hospital and Loyola University School of Medicine, Chicago, and Dr. John D. Claridge, assistant professor of orthopedics at Loyola, and a member of the staff of Mercy Hospital, Chicago. Dr. Smith held a clinic open to all members of the profession at the Natchez Charity Hospital for Dr. Christopher.

E. R. GORDON.

It is with much regret that we announce the death of Dr. E. R. Gordon of McComb. Dr. I. E. Stennis, Secretary of the Pike County Medical Society, furnishes the following:

"Dr. E. R. Gordon, head of the McComb Infirmary, and one of the best known physicians in South Mississippi, died at 7:30 o'clock, Tuesday morning, February 18, of heart and kidney trouble.

"Dr. Gordon was born in Amite County, Miss., fifty-two years ago, moving to McComb with his parents, Dr. and Mrs. T. J. Gordon, and becoming associated with his father in the drug business, and also in the practice of medicine. He was a leader in social and professional life in McComb, having practiced medicine 26 years.

"Dr. Gordon studied medicine at Tulane University and later at Louisville, Ky. During the World War he served as captain in the medical corps of the 42nd Division. Most of the time during the war was spent on the front, and he was gassed while in service, from which it is thought he never recovered. A great deal of his time of late years had been devoted to his Infirmary, and an addition was in course of construction, which he was not spared to see completed.

"Funeral services were conducted at the First Baptist Church, of which church he was one of the most devoted and helpful members. Services were conducted by Dr. J. W. Mayfield, his pastor; Rev. H. L. Carter, of the Central Baptist Church, with whom he served in the war; Rev. J. H. Lane and Rev. Elisha Gardner of Mars Hill, and under the auspices of the Knights Templar.

"Dr. Gordon was married about twenty-five years ago to Miss Julia Ellzey, who with a little daughter, Louise, an only sister, Miss Mae Gordon, a brother L. E. Gordon, all of McComb and two nieces, Miss Mary Gordon of McComb and Miss Nellie Gordon of New Orleans, survive him.

"Dr. Gordon was a member of the Knights Templar, American Legion, and many civic clubs in McComb. The many floral offerings attested the high esteem in which he was held."

FROM ANOTHER WHO KNEW DR. GORDON.

"It was my good fortune to know E. R. Gordon fairly well. During the early winter of 1917-18 we were at the Base Hospital at Camp Logan and, being the only Mississippians there at that time, drew close together. So when I met him next, at the meeting of the State Medical Association in Hattiesburg in 1919, it was but natural that I, who had been one of the unfortunates who stayed at home, should at once demand to know what he had done and how he had fared, and this was his story, in almost these very words: 'You know I left Logan in charge of a bunch of corps men, we went across at once and got there in time to get mixed up in the drive the Germans made to capture the Channel Ports (March 1918). Then I was sent down into France and assigned to a machine gun battalion, and went with them through the Argonne and on into Germany with the army of occupation.'

"Short and to the point, and typically Gordon. He might have been telling of a vacation trip down on the Gulf Coast and over into Alabama, only had he been doing so, he would probably have had something to say about the scenery along the road or the places he visited.

"But I learned more about it the next day. We had been out to Camp Shelby, to the meeting the Association held there the second day, and were at the Camp Station waiting for a train back to Hattiesburg, when along came a group of young officers. I learned later that they were from some of our Western States and had just been discharged from the army at Shelby. One of them glanced in our direction and let out a yell. The others instantly followed suit, and then they came at Gordon.

"They picked him up and hugged him. They sat him down and danced around him. They passed him from one to another and patted him on the back and hugged him some more. I didn't see any of them actually kiss him, but I have no doubts they did. Then the train came along and they picked him up and put him aboard, but here came a hitch, because they all wanted to sit with him. So they compromised by turning two seats together so they could sit facing each other and took turns holding him in their laps all the way to Hattiesburg.

"But just before we left the train I caught one of them and asked what it was all about. The reply was too incoherant for me to quote verbatim, but it was about like this: 'Why, By God, Sir, that's the finest fellow that ever lived! Our battalion never would have gone through the Argonne if it hadn't been for him! You'd see him walking all day so a couple of chaps with sore feet could ride; and he'd be up half the night looking after the fellows and making them comfortable, and patching them up so they could keep on going, and when anybody was hurt he was always right there, right on the job! Why, when he got the flu in Germany and was sent back, all the battalion sat down and cried. We were just lost without him.' And the young fellow pulled out his handkerchief and openly wiped his eyes, not caring in the least that everybody around should see he was doing some more crying.

"I don't know what sort of an obituary will be written. Except for the good it will do as an example, none is needed, for he wrote it himself in the memories of those young men, and it will stand out bold and clear whenever they think of Gordon."

Dr. Guy R. Jones, formerly house surgeon at Hotel Dieu, has acceptel a position as assistant to Dr. D. E. Martin of the Martin Sanatorium, Picayune.

VICKSBURG SANITARIUM

The regular monthly meeting of the Staff of the Vicksburg Sanitarium and Crawford Street Hospital was held on April 10. Special case reports presented included:

1. Carcinoma of the Appendix.—Dr. G. M. Street.

2. Liver Abscess.—Dr. A. Street.

3. Infection of the Eye Simulating Gonorrheal Opthalmia.—Dr. C. J. Edwards.

Dr. J. A. K. Birchett, Jr., presented a special report on the use of Spinal Anesthesis in Abdominal Surgery, and Dr. E. H. Jones a special report on the effect of Certain Drugs on the Nasal Mucosa.

Selected Radiographic Studies were shown as follows: Pulmonary Tuberculosis (2 cases); Osteomyelitis of the Femur; Cholelithnasis; Fracture-dislocation of the Neck.

The Pathologist reported for the month sixteen malignant growths as follows: Adeno-carcinoma of the Appendix (2); Adeno-carcinoma of the Breast (3); Adeno-carcinoma of the Rectum (1); Adeno-carcinoma of the Stomach (1); Adeno-carcinoma of the Uterus (1); Carcinoma of the Thyroid (1); Squamous cell carcinoma of the Cervix Uteri (4); Carcinoma of the Skin of the Face (3). Non-malignant growths for the month were reported as follows: Fibro-adenoma of the Cervex Uteri (2); Fibro-adenoma of the breast (1); Cystoma of the Ovary (7); Fibro-lipoma of legs and buttocks (1); Lipoma, subcutaneous (3); Adeno-cystoma of Penis (1); Fibro-leiomyoma of Cervix Uteri (1); Fibro-leiomyoma of Uterus (2); Fibroma of Breast (1); Fibroma of Axilla (1); Retention cyst, subcutaneous (1).

Drs. Baysinger and Crawford of Jackson were visitors.

BOOK REVIEWS

Roentgenographic Technique: By Darmon Artelle Rhinehart, A. M.-M. D. Philadelphia, Lea & Febiger. 1930. Pp. 388.

This manual is intended for physicians, students and technicians.

A very comprehensive explanation is given of the modern roentgen-ray technic, without diagnostic interpretation. It is profusely illustrated demonstrating the different positions used in making skiagrams of the human body.

About one-half of the book is taken up in describing electric apparatus, developing technic etc., and the other half is devoted to roentgen ray technic.

This book will be found useful to technicians as it contains the most modern methods used at the present time in making skiagraphic examinations.

LEON J. MENVILLE, M. D.

Clinical Atlas of Blood Diseases: By A. Piney, M. D., M. R. C. P., and Stanley Wyard, M. D., M. R. C. P. Philadelphia, P. Blakiston's Son & Co. Inc. 1930. Pp. 99.

A collection of some 36 plates illustration various types of blood disorders, with a brief synopsis of the general findings of the disease illustrated.

J. H. MUSSER, M. D.

Insomnia—How to Combat It: By Joseph Collins, M. D. New York, D. Appleton and Company. 1930. Pp. 130.

Any writings from the pen of Joseph Collins may be safely assumed to be written with charm and with delightful English of a remarkable essayist. In addition to the excellent presentation, the subject matter is lucid, sensible and equitable.

J. H. MUSSER, M. D.

Orthopedic Surgery: By Sir Robert Jones, Bart., K. B. E., C. B. and Robert W. Lovett, M. D., F. A. C. S. 2nd ed. rev. New York, William Wood and Company. 1929. Pp. 807.

All specialists in this branch of surgery as well as all interested in bone and joint surgery know of and possibly possess a copy of the first edition of this work published in 1925 by the deans of orthopedics in this country and England.

This, the second edition, has been revised in its entirety, and to it has been added chapters on the principals of treatment of fractures at all stages; (2) a chapter on the affection of tendons, muscles and fascia; (3) a chapter on peripheral nerve lesions; (4) one on pyogenic infections of bone; (5) one on amputations and artificial limbs. These additions, together with the revisions, makes this volume a complete, up-to-date treatise covering all conditions seen by the orthopedic surgeon.

H. THEODORE SIMON, M. D.

Essentials of Histology: By Sir Edward Schaffer, (Twelfth edition, revised by the author with the assistance of Dr. H. M. Carleton). Philadelphia, Lea & Febiger. 1929. Pp. x+628, 758. figs.

Since the first appearance of this text-book of histology and organology, in 1885, revisions have been made at fairly close intervals, the work being now available in the enlarged twelfth edition. The succession of editions in itself signifies recognized merit. The present revision contains much new material, both in text and illustrations. It sustains the well-earned reputation of the book for clear style of presentation, judgment in choice of material and abundance of instructive figures.

HAROLD CUMMINS, Ph. D.

A Textbook of Orthopedic Nursing: By Evelyn C. Pearse; foreword by Sir Robert Jones, Bart., K. B. E., C. B., F. R. C. S., and an introductory chapter by Dame Agnes Hunt, D. B. E., R. R. C. New York, G. P. Putnam's Sons. 1930. Pp. 155.

The technic of orthopedic nursing offers many difficulties to the nurse who is unfamiliar with this class of patients. Evelyn C. Pearse, with a background of many years of experience in this work has covered the field very well in her unique volume. The congenital and acquired deformities are carefully explained, with a brief outline of the treatment, and an excellent treatise on the care of the particular case make her book a valuable addition to the library of the nurse. Anatomy and function of every joint is stressed in the making of plaster bandages, the preparation of the part for operation and for the application of casts, the fitting of splints, and the care of the patient following the application of casts.

DUDLEY M. STEWART, M. D.

The Newer Knowledge of Nutrition: By E. V. McCollum, Ph. D., Sc. D. & Nina Simmonds, Sc. D. New York, Macmillan Co. 1929. Pp. 594.

This is a new edition of a standard text that has been extensively revised. The work is authroitative and exhaustive and yet brief and to the point. An excellent bibliography is attached.

I. L. ROBBINS, M. D.

Research and Medical Progress and Other Addresses By J. Shelton Horsley, M. D. St. Louis, C. V. Mosby Co. 1929. Pp. 208.

This volume is a collection of many excellent addresses that have appeared in many of the medical Journals. The author is well known and anything by him may be read with interest and profit.

I. L. ROBBINS, M. D.

Surgery at the New York Hospital One Hundred Years Ago: By Eugene H. Pool and Frank J. McGowan. New York, Paul B. Hoeber, Inc. 1930. Pp. 188.

A charming little account of surgery one hundred years ago, printed with the usual skill and artistry of Hoeber.

J. H. MUSSER, M. D.

Aids to Orthopedic Surgery: By Eric A. Crook, M. Ch. (OxOn). F. R. C. S. (Eng.). New York, Wm. Wood & Co. 1929. Pp. 232.

This is a very interesting and well written treatise which as the author says in his preface: he has endeavored to cover that part of Orthopedic Surgery which is required of the student in general surgery.

There are chapters on congenital deformities, traumatic conditions such as fractures, joint wounds, neuro-muscular injuries, etc. Also on static conditions, inflammatory conditions and new growths. The author goes into all of these things very briefly and states only the salient points in the symptoms, diagnosis and treatment.

There is a short paragraph on the ossification of the epiphyses.

The book will undoubtedly be appreciated by the general practitioner.

EDWARD S. HATCH, M. D.

A Study of Masturbation and the Psychosexual Life: By John F. W. Meagher, M. D., F. A. C. P. 2nd ed. New York, Wm. Wood & Co. 1929. Pp. 130.

A Study of Masturbation, by John F. W. Meagher, is a very brief, but comprehensive, treatise not only on masturbation in its broad sense, but on the psychology of sex and sexual development, and should be a valuable edition to the library of any one interested in these matters. Particularly people who are dealing with mental hygiene in its various fields will find it an excellent book to familiarize themselves with. Its language is simple and it is written in such a manner that the training of a specialist is not required for its assimilation. The family physician, ministers, priests, social workers—any one, in short, coming in contact with problems of human behavior and human adjustment—should appreciate a book of this type as giving them an insight into a most important angle of their work.

E. McC. CONNELY, M. D.

Bulletin of the National Research Council: A Survey of the Law Concerning Dead Human Bodies: By George H. Weinmann. Washington, D. C., National Research Council. 1929. Pp. 199.

A compound of the laws of different states dealing with such subjects as the "dead body;" property rights; coroners and coroners' inquests; autopsies; disposition; exhumation or disinterment. A particularly valuable summary for those who come in contact with forensic medicine.

J. H. MUSSER, M. D.

A Textbook of Physiology for Nurses: By William Gay Christian, M. D. and Charles C. Haskell, B. A., M. D. St. Louis, C. V. Mosby Company. 1929. Pp. 153.

An excellent presentation concerning the functions of the body which is very clearly written with a minimum amount of detail. Probably the only criticism that might be made is that too much space has been allotted to the nervous system and the special senses in comparison with the space allotted to other subjects.

J. H. MUSSER, M. D.

Minor Surgery: By Frederick Christopher, M. D., F. A. C. S., with a foreword by Allen B. Kanavel, M. D., F. A. C. S. Philadelphia, W. B. Saunders Co., 1929. pp. 694.

There has been a real need for a good text of minor surgery for several years and it is with real pleasure that I report its arrival.

Christopher has given us a comprehensive work with many excellent features and no serious omissions.

Diagnosis, pathology, and treatment are fully considered and in a fashion so clear and practical that the student has a good working knowledge of each subject without the necessity of referring to other books.

Surgical handicraft is admirably discussed. Practical technical suggestions are numerous and will repay any experienced surgeon for the time spent in study of the volume.

The fundamental pathology and physiology on which treatment and diagnosis are based will be found fully and clearly described.

In short this is an excellent book deserving a place in every good surgical library.

Only a disagreeable tendency to criticism prompts the reviewer to make one unfavorable comment. It is certain that the author does not use nor approve the use of the many methods of treatment detailed. A more diagnostic attitude would be better for medical students. American authors appear to have a fear of expressing definite opinions on controversial subjects that amounts almost to a vice.

J. D. RIVES, M. D.

Pathogenic Microorganisms: By William Hallock Park, M. D., Anna Wessels Williams, M. D., and Charles Krumwiede, M. D. 9th Ed., enl. and rev. Philadelphia, Lea & Febiger. 1929. pp. 819.

This book should be of great practical interest to the clinician, as well as the laboratory worker and student. An excellent volume, concise, comprehensive and unlike most books on bacteriology which are for laboratory workers only.

Inquiry in the field of bacteriology is so active at the present day that no general text-book can maintain its usefulness long without frequent revisions. It has been gratifying to note how much of the work which seemed to us particularly valuable and enlightening has emanated from American laboratories.

Experiences with active immunization against diphtheria and scarlet fever not only are the immediate practical results emphasized but, also, those observations which bear on the general problems of immunity, also to the value of certain serums and vaccines.

Many lengthy and unnecessary details having been condensed to a few paragraphs. The chapter on compliment fixation has been revised. Details regarding the newer precipitation tests, especially those of Kahn, Kline and Meinike are added. Results of the use of the feeding the Calmette Vaccine to infants. However, the authors are beginning to vaccinate the older infants with small subcutaneous or intracutaneous injections, giving from one-hundredth to one-tenth milligram.

Special effort has been made to give those references which cover the essential advances during the last few years in penumonia, measles, scarlet fever, etc.

J. L. LOCASCIO, M. D.

Otologic Surgery: By Samuel J. Kopetzky, M. D., F. A. C. S. 2d Ed., rev. New York, Paul B. Hoeber, Inc. 1929. pp. 553.

In this work Dr. Kopetzky has covered the operative surgery of the ear and the mastoid, and the operative surgery of intracranial and blood stream infections secondary to aural diseases. The text is clarified by the use of 104 well selected illustrations and 21 charts.

Dr. Kopetzky calls our attention to an interesting point when he discusses the dangers of too early incision of the ear drum. He says, "As the result of long observation it has become evident that to incise the drum too early in the course of the development of an acute suppuration of the middle ear is to add a factor toward the promotion, rather than toward the retardation, of the middle ear infection."

The volume will be found an extremely valuable reference work for the otologist.

H. KEARNEY, M. D.

PUBLICATIONS RECEIVED.

The Williams and Wilkins Company, Baltimore: Immunity in Infectious Diseases, by A. Besredka, authorized translation by Herbert Child, N. R. C. S. (Eng.), L. S. A.

P. Blakiston's Son & Co. Inc., Philadelphia: Medical Gymnastics and Massage in General Practice, by Doctor J. Arvedson, translated and edited by Mina L. Dobbie, M. D., B. Ch.

Paul B. Hoeber, Inc., New York: Human Biology and Racial Welfare, edited by Edmund V. Cowdry.

C. V. Mosby Company, St. Louis: The Normal Diet, by W. D. Sansum, M. S., M. D., F. A. C. P.

F. A. Davis Company, Philadelphia: Normal Facts in Diagnosis, by M. Coleman Harris, M. D. and Benjamin Finesilver, M. D. Varicose Veins, by H. O. McPheeters, M. D., F. A. C. S. Trauma, Disease, Compensation, by A. J. Fraser, M. D. Venereal Disease, by Hugh Wansey Bayly, M. C. Modern Otology, by Joseph Clarence Keeler, M. D., F. A. C. S.

Harper & Brothers, New York and London: Uterine Tumors, Charles C. Norris, M. D. Cancer of the Breast, by William Crawford White, M. D., F. A. C. S.

The Lister Medical Press, St. Louis: The Treatment of Skin Diseases, by Noxon Toomey, M. D., B. A., F. A. C. P.

The Oboschell Corporation, New York: Lectures on Colonic Therapy. by Boto Schellberg.

Gaston Dion & Cie. Paris: Les Syndromes Ictériques, by P. A. Carrie.